THE
VETERINARY JOURNAL

Editor:
FREDERICK HOBDAY, C.M.G., F.R.C.V.S., F.R.S.E.,
HONORARY VETERINARY SURGEON TO HIS MAJESTY THE KING,
OFFICIER DU MÉRITE AGRICOLE (FRANCE),
CAVALIERE DEI SS. MAURIZIO E LAZZARO (ITALY),
HONORARY MEMBER OF THE AMERICAN VETERINARY ASSOCIATION,
PRINCIPAL OF THE ROYAL VETERINARY COLLEGE, LONDON.

Sub-Editor:
GLADSTONE MAYALL, M.R.C.V.S.

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POULTRY DISEASES AND VETERINARY RESEARCH.

IT is only during comparatively recent years that the members of the veterinary profession have seriously considered poultry diseases as being worthy of their attention, but when one is informed that there is more money invested in poultry breeding in Great Britain than there is in the production of wheat, one realises that there are great possibilities for our graduates; and that, if properly approached, there are plenty of opportunities for research in a new field of work. It is not merely to be looked upon by the practitioner as an *additional* means of adding to one's livelihood, but to the laboratory scientist the study of poultry diseases affords a most varied and fascinating field of work, not only amongst the bacterial and invisible virus infections, but also amongst tumour formations and such like things, which the uninitiated have imagined as belonging solely to animal patients.

It was not until after the Great War that serious attention was paid by veterinary scientists in Great Britain to poultry diseases. Up to that time any person who wished to know the causes of deaths in fowls or other birds knew of no better place to which to send a body for post-mortem examination than the office of a poultry paper, where (usually for a minimum of 2*s.* 6*d.*) it was examined by a so-called expert, and an opinion given either direct to the sender of the body, or in the next week's column of replies to correspondence. Occasionally illnesses were diagnosed, and remedies prescribed, upon the descriptions of symptoms shown, these replies also being given by "ready writers" whose scientific and professional qualifications were *nil*, or at best dubious.

Other European countries have for many years included the diseases of poultry and other birds in the curricula of their respective Veterinary Colleges, and in America this also applies.

In Great Britain the credit for giving a serious lead on this most important question must be given to the Ministry of Agriculture (probably because, after

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the War, so many ex-soldiers put their hard-earned gratuity money into chicken farming), who handed it over to the Veterinary Department, in whose well-equipped laboratory at Weybridge some excellent pioneer work has been done. Certain of the Universities, especially those of Cambridge, Edinburgh and Aberdeen, the Wellcome Laboratory, and certain of the Agricultural Colleges (especially the Harper Adams) have also seriously joined in; and some three years ago a Canadian veterinary graduate, Dr. Patrick Hamilton (B.V.Sc. Toronto) started in a small way a private poultry research laboratory, mainly with the idea of assisting the fowl owners to eradicate from their flocks that most fatal and insidious disease which is termed for brevity's sake, " B.W.D." The need which there was, and the success which has attended his effort, is shown in the article on page 5, and by the fact that in 1931 more than a million samples of fowls' blood had been examined in this laboratory alone for this one disease. Truly a pioneer result, which should stimulate further efforts on behalf of other veterinary workers to take up poultry disease research.

Quite recently, too, the International Poultry Congress was held at Hanover, the meeting being an outstanding success; and, although the paucity of the number of delegates who attended from this country was deplorable, our foreign colleagues gave a good account of poultry research work, with much promise for the future.

Our effort in endeavouring to produce a Special Poultry Number of THE VETERINARY JOURNAL, the second issue devoted within eighteen months to this subject, will show that the matter is not completely neglected in this country, and that the British veterinarian is at last awakening to its importance. It is a branch of the profession which is admirably suited for the women graduates, and we commend it to the notice of those who intend to adopt a laboratory career as worth serious consideration both from the scientific aspect and from the ever necessary aspect which we all have to consider, *i.e.*, as a satisfactory means of earning a livelihood.

DISTEMPER IMMUNISATION.

WE call the attention of our readers to two important letters on pages 46 and 47, dealing with the history of immunisation against distemper, and the use of dried virus, as they explain away any erroneous impression which the Editorial in the December issue may have unintentionally conveyed.

THE JOURNAL IN 1932.

IT will be noticed that in this issue THE VETERINARY JOURNAL takes on a new dress. The old long and narrow page has given place to the much pleasanter broader and squarer one. Technically the change is from a royal octavo to a crown quarto size. We feel sure that the alteration is all to the good, as it makes

the Journal more attractive in appearance and easier to read and handle, for lettering on shiny paper is much more tiring to the eyes than that on a rough surface.

It cannot be said that the publishers are running after the latest fashion, for we find on referring to the files that it was in the year 1900 that the last change in *format* was made ; the editorial chair being then occupied by Lieut.-Colonel J. A. Nunn. Many improvements have been made in the Journal since then, the most marked being in relation to the reproduction of photographs and other illustrations, but now, in this our twenty-eighth year of office, the publishers have made this radical change from its old familiar appearance, thus bringing the Journal into line with modern ideas of typographical form. We welcome the changes, and feel sure our readers will also. *Never was there a time when the study of veterinary science in every field was more important than now, nor when there were more problems to be solved ; and never were there fewer men willing to put their hands to the plough.* We need, as a profession, to arouse ourselves, and to be prepared for the strenuous time which the profession has in front, not only in supplying the ever-increasing needs of the Empire in efforts to create an efficient preventive service against animal epidemics, but to train men to undertake the ever necessary duties of the general practitioner, who is, after all, the backbone of the whole organisation.

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General Articles

WHAT THE POULTRY INDUSTRY NEEDS FROM THE VETERINARY PROFESSION.

By C. A. HOUSE,
Managing Editor of "Poultry World."

In this country veterinary science is severely handicapped by reason of the fact that our Government gives little help to the profession to carry out research work, and as the teaching colleges are not over-burdened with funds, they are compelled to devote their time principally to the larger animals. All who have any knowledge of the matter know the truth of this. At present the students, having gone through their course at the College, go out to practice with little but theoretical knowledge concerning these things. It is necessary that students of the future should go out to their life's work with knowledge that will fit them to deal with the diseases of birds; and it is necessary to remove the reproach which all too long has existed, that our veterinary surgeons know little about the poultry industry and what it requires from the profession. In a number of countries abroad, thanks to Government assistance, many veterinary surgeons specialise in the diseases of fowls and other birds. Why should it not be so in this country?

One very important thing that is needed is a closer study of the physiology of the fowl. This acquired, the fundamental facts governing the diseases which trouble the keepers of poultry will be the more readily realised, and our veterinary surgeons will get nearer to the respective causes of poultry ailments. When causes have been discovered, treatment and prevention will follow.

Women are taking up veterinary work. The avian diseases afford them scope for being specialists. They should grasp it with both hands. The entrance of women into the profession should alter the manner in which this subject of avian disease has been approached in the past. We grumble at our "vets.," and say it is because of the bigger fees paid in other fields of the profession. True! but we must remember that men must live. The small fees paid, apart from the lack of college training, have led to neglect in the past, and breeders have found it cheaper to kill affected birds than to pay an amount which would adequately recompense individual veterinary surgeons for the time and skill given in their treatment.

In the veterinary world, as in the medical, far more attention has been given to the incidence of disease than to its prevention. One of the most pressing things which the poultry industry requires of our veterinary profession is co-operation between laboratory workers and practical workers in the field; and although laboratory work may take the students away from the theories of the

past, it will not give them the *field practice* which is needed before they can give the poultry industry much practical help.

It is only when the results of laboratory research can be satisfactorily applied by the poultry farmer that they can be of much service to him.

At the moment, with the widespread devastation caused by such diseases as coccidiosis and fowl paralysis (if this is a disease apart by itself), prevention, even more than cure, is what is needed. If matters could be arranged so that the Royal Veterinary College could work in collaboration with the National Poultry Council, it might be possible to form a very strong practical consultative committee which would be of great value and help to poultry owners whose flocks are at the present time devastated by parasitic and other epidemics. I give these suggestions to the profession for serious consideration, and, as my part, I would gladly lend what help I could from the poultry press.

A POULTRY PATHOLOGICAL RESEARCH LABORATORY.

By H. P. HAMILTON, B.V.Sc. (Toronto).

Goring, Reading.

DURING the last year of my course in veterinary science at the Ontario Veterinary College, Guelph, Canada, I decided to specialise in the study of poultry disease, and started a poultry disease laboratory in that town after obtaining my B.V.Sc. degree from the University of Toronto. On my return to England from Canada in the autumn of 1928, I made inquiries regarding the position of poultry disease in this country. The result of my investigations led me to believe that there was room in the industry for such a laboratory, which would be devoted solely to the diagnosis of poultry disease, and to the application of the blood serum agglutination test for pullorum infection in adults. My suggestions met with the warmest approval of numerous county poultry instructors and eminent breeders. It was with this knowledge and confidence behind me that I decided to start the venture.

All the buildings are of wooden sectional construction, and were built by the disabled soldiers under the Village Settlement Scheme at Enham, Andover. The organisation at present in force falls into five main headings, *viz.*, serological, under the personal control of myself as Director; post-mortem examination, under the control of Mr. Axworthy, N.D.P.; chemical analysis, under Miss King, D.Sc.; physiological research, under Mr. W. P. Blount, F.R.C.V.S.; and secretarial, under Mr. N. W. Kirkby, B.A.

In the serological department, more than one and a quarter million samples of the blood from adult poultry have been examined for *Salmonella pullorum* infection during the past year, and some twenty-five persons are employed in this department alone; whilst from our records we are able to state an accuracy of

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approximately 97 per cent. An important branch of this department is the liaison service, which consists of a responsible representative visiting farms where the agglutination test has been applied, and ascertaining the results obtained in hatching and rearing chicks. This constitutes a most important link between the laboratory and the field.

In the post-mortem department many thousands of birds, dead from all manner of diseases, are examined annually, and the same applies in a lesser degree to the chemical and analytical department, where samples of food are examined for protein, carbohydrate, ash, water and fibre content. Animal tissues and organ contents are also examined for the common poisons.

The physiological research department is to be of considerable value to the poultry industry. Mr. Blount is now working on the ætiology of so-called " fowl paralysis," and hopes to enlist the co-operation of fellow veterinary surgeons and medical men in his attempts to elucidate this very difficult problem. The respective incidences of coccidiosis and helminthism are also receiving attention.

The secretarial department, always a most important one in any business undertaking, is self-explanatory.

The new buildings were opened on July 23rd, 1931, by Sir Edward Brown, Secretary of the National Poultry Council, and the proceedings were attended by Dr. te Hennepe (who came from Rotterdam especially for the occasion), and by Mr. G. P. Male, M.R.C.V.S., of Reading, the President of the Royal College of Veterinary Surgeons.

As an old student of the Royal Veterinary College, and wishing to show some small appreciation of the help received from this Institution, and especially from its Principal, I have decided to make a contribution to the College funds amounting to one-twentieth of a penny per agglutination test carried out in the laboratory. It is hoped that the money so received will be used by the College towards the salary of a full-time research worker and lecturer in poultry diseases.

Old students of the London College will be interested to know that Mr. A. A. MacKay, who for so many years was on the laboratory staff, is installed here as senior assistant in the serological department, where he is busily engaged in manufacturing the biological products necessary in the carrying out of the serological tests. These products include *S. pullorum* and *E. sanguinaria* antigens, and tuberculin for the diagnosis of avian tuberculosis.

The laboratory staff at present numbers fifty-five persons, all exclusively engaged in work connected with the diagnosis of poultry disease. Veterinary surgeons can confidently consult us regarding the ailments of birds belonging to their clients, knowing that the case in point will receive the attention of specialists.

Two years' concentrated work on the diseases of poultry clearly shows one outstanding fact. The physiology of the subject *must* receive greater attention. It is not the *cure* of poultry diseases that matters, but the *prevention* of their occurrence ; and in any scheme for prevention of disease, physiology plays a most important part. Readers of veterinary literature will have realised that

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A View of the Laboratory

"A Poultry Pathological Research Laboratory," by H. P. Hamilton, B.V.Sc. (Toronto), Director. (*Page* 6.)

Fowl Suffering from Pericarditis

Note enormously enlarged pericardial sac which contained a large quantity of sero-fibrous exudate.

"Pericarditis of the Fowl," by K. D. Downham, B.V.Sc., M.R.C.V.S., D.V.H. (*Page* 44.)

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Mr. W. P. Blount is a physiologist, and as such he will have ample scope for the extension of his interests. Too much attention has been given of late years to attempting to cure such diseases as fowl pox, coccidiosis, paralysis, etc., but very little real success has been obtained. When dealing with the fowl, which the poultry breeder has developed into a veritable egg machine, and with modern rearing methods such as battery brooders, etc., extra care must be taken to maintain the stamina of the bird, and not to allow any particular organ to suffer a reduction in its physiological function. There is an old saying: "we lose that which we abuse or do not use," and this distinctly applies in a particular way to the modern domestic fowl.

OBSERVATIONS UPON SOME PREVALENT DISEASES OF FOWLS.

By PROFESSOR D. C. MATHESON,

Department of Poultry Diseases, Royal (Dick) Veterinary College, Edinburgh.

THE present article forms a contribution to a wider investigation upon which the writer is engaged from the epizoötiological point of view into the prevailing diseases of poultry. As pointed out elsewhere (Matheson[1]), the writer has set himself four questions: (1) Which are the prevailing diseases of Poultry? (2) Why are they prevalent? (3) What can be done to reduce their prevalence? (4) What can be done to prevent their occurrence?

The purpose of the present article is to provide some original data towards an answer to the first question. In a report presented to the Third World's Poultry Congress, the writer (Matheson[2]) published a list, based upon the records of his laboratory over a period of thirteen years, of the prevailing disease of British fowls. More recently he has been engaged upon an analysis of the records of six high-grade flocks, each of which has been under his special observation for a year. The results of that analysis will be published elsewhere (Matheson[3]), but it is interesting to notice that the five diseases found to be prevalent in these flocks, in the sense that they occurred during each year of observation, and therefore in each flock, although the number of cases was small, as the health of the flocks was good, also appear in the earlier list. The first list was based upon examinations of a number of fowls of all grades and varying ages received from time to time over a long period; the second list, below, upon the examination of six high-grade flocks coming under observation as pullets, and remaining under observation for one year. The five diseases which appear in each list are: (1) Tuberculosis; (2) impaction of some portion of the alimentary tract, usually "grass-ball" in the crop; (3) tumours; (4) fatty degeneration of the liver, often with a terminal hæmorrhage; (5) impaction of the oviduct.

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PREVAILING DISEASES AMONG SIX HIGH-GRADE FLOCKS.

Disease.	Total number of cases.	Average number each year	Percent. of the total number of deaths.	Percent. of the population.
Tumours	28	4.6	12.1	0.8
Tuberculosis	22	3.6	9.5	0.6
Impaction of the Crop ...	20	3.3	8.6	0.6
Impaction of the Oviduct ...	17	2.8	7.3	0.5
Fatty degeneration of the Liver	12	2	5.2	0.3

Calculations are carried only to one place of decimals.

We may now briefly consider the above table.

Tumours.—For the purpose of the present discussion, the malignant and the so-called benign growths are grouped together, and, thus taken, constitute the major source of loss among these flocks.

Tuberculosis.—Tuberculosis in this connection has already received attention (*see* VETERINARY JOURNAL, January, 1930), but it may be noticed that it is the only specific disease at present associated with a micro-organism to appear among the prevalent diseases as above defined of these high-grade flocks.

Impaction of some portion of the alimentary tract, usually impaction of the crop, "crop-bound," "grass-ball."—There are several forms of impaction of the crop met with among poultry: the form to be discussed here is that associated with the presence of a "grass-ball." Manipulation of the crop suggests, what the post-mortem examination usually confirms, that a larger or smaller mass of tangled grass stalks, straw, hay, bristles, or feathers is present in the crop, and sometimes continued down for some distance into the second portion of the œsophagus as a veritable rope. The condition has been specially studied in connection with the six flocks mentioned above. The fowls in these flocks are arranged in groups of six, each group has a separate house and grass run; this accommodation is referred to as a "pen." The birds in any year are fed alike. The condition of impaction or of "grass-ball," as described above, is considered at the moment to be due to a depraved appetite associated with indigestion: this leads the bird to eat the grass stalks, etc., which it cannot subsequently digest. This consideration is supported by: (1) The food supplied was adequate, since the pen mates of the affected bird throve and laid eggs. (2) Only one case occurred in any pen during the year of observation. (3) Cases were most frequent between the middle of November and the middle of December, and again from the middle of June to the middle of July; that suggests the condition of the grass had nothing directly to do with the cases. (4) The other birds in the pen had equal opportunities of eating the grass stalks had they so desired; examination of the crops of birds dying from other causes failed to show any accumulation of grass stalks, etc., such as might has given rise to impaction or "grass-ball" had the birds survived.

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Impaction of the Oviduct.—By impaction of the oviduct in this connection, the writer does not mean the relatively simple condition of obstruction of the oviduct, " egg-bound," as it is commonly called, associated with the presence of an extra large or malformed or misplaced egg which the hen may experience difficulty in expelling from the oviduct, but a condition in which a quantity of egg material, exudate, cell debris, etc., accumulates in the oviduct over a period—it may be a relatively long period—until a massive impaction results ; the abdominal cavity becoming filled up by the enormously distended oviduct, and the viscera pressed upon, and even partially displaced. As might be expected, these cases tend to occur towards the close of the period of observation, which means that the accumulating material is reaching the limit of the bird's tolerance for it.

Fatty Degeneration of the Liver.—Among the domesticated animals, fowls appear peculiarly liable to rupture of the liver, and to consequent death from hæmorrhage. Fatty degeneration of the liver—one of the prevailing diseases—disposes to such rupture and hæmorrhage. Rupture of the liver is also not uncommon in the fowl affected with tuberculosis. Fatty degeneration of the liver, often with a terminal hæmorrhage, accounted for 5.2 per cent. of the total deaths among the flocks at present under consideration. Cases tended to occur towards the latter half of the period of observation ; the birds were fed alike, yet there was not more than one case of the disease in any one pen in any one year ; it would appear as if the constitution of the affected birds were mainly responsible for the condition.

Acknowledgments are due to Mr. J. E. Wilson, B.Sc., M.R.C.V.S., and to Mr. Alex. Ratter, M.R.C.V.S., of the above department, for assistance in connection with the post-mortem examinations.

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A HÆMOGLOBINOPHILIC BACTERIUM AS THE CAUSE OF CONTAGIOUS CATARRH OF THE FOWL.

(Coryza infectiosa gallinarum.)

By Dr. L. DE BLIECK,
Utrecht, Holland.

In a report issued to the World Poultry Congress at Ottawa in 1927, I took the view that contagious catarrh in poultry was a distinct disease. The same view had already been adopted by Beach, Hareng, and Kofoid. In a great number of cases of chickens recovered from diphtheria or fowl-pox, it was possible to infect them with contagious catarrh, and *vice versa*. Both diseases, however,

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often appear together as a mixed infection, one acting as a suitable predisposant for the other. Usually both appear in the autumn and the beginning of winter.

Van Heelsbergen has also drawn attention to the necessity of distinguishing infectious or contagious catarrh and diphtheria as separate diseases. It is difficult to adopt this view as long as one does not understand the actual cause of contagious catarrh. Doyle and Minett state that they are able to demonstrate fowl-pox virus in clinically uncomplicated infectious catarrh. They do not, however, go so far as to say that fowl-pox virus is the cause of contagious catarrh, but rather that it is an accompaniment of this malady. In his manual, Hutyra remarks that in young chickens fowl-pox diphtheria can manifest the symptoms (nasal discharge) of contagious catarrh. Some experiments I carried out in this direction proved to me that when fowl-pox virus was injected into the nostrils of young chickens it gave rise in some cases to a catarrhal inflammation which began with a sero-purulent discharge, but was soon followed by the diphtheritic process. On the contrary, I have never been successful in producing fowl-pox diphtheria with the nasal mucus of uncomplicated contagious catarrh.

Since 1929, I have been endeavouring to transmit infection from fowl to fowl with the virus of contagious catarrh. For these experiments I used fowls that had been rendered immune to fowl-pox : diphtheria with anti-diphtherin, and in consequence of this I have been successful. Starting with a case of clinically uncomplicated contagious catarrh, that is to say, one with a nasal muco-purulent discharge without any diphtheritic or croupous membranes, I succeeded in constantly producing the selfsame symptoms of the disease in about 200 birds, the manifestations being conjunctivitis and the above-mentioned nasal discharge, in addition to the appearance at intervals of œdema round the eyes, and which in some instances extended as far as the throat. These fowls were kept under observation from between six weeks to eight months without any diphtheritic membranes appearing.

Starting from a case of mixed infection fowl-pox + diphtheria + contagious catarrh, I succeeded in separating both diseases in birds that were immunised against fowl-pox + diphtheria by anti-diphtherin. With the contagious catarrh virus obtained in this manner, further cultivation was carried out on a great number of fowls. Further information in connection with these experiments, and of the details that were observed, will be published later on. It has become evident that when infection is transmitted by the nasal mucus from one animal to the other, the infection amounted to 100 per cent. in those young birds that had not previously suffered from infectious catarrh. It is not of much importance whether the nasal secretion is inoculated during the first stages of the malady, or it takes place a month, or even later, after the first signs of the phenomenon. I also succeeded in transmitting this disease by contact. The results, however, were not always constant, especially when only one bird was placed with a single affected bird. Taking one thing with another into consideration, I became convinced that this experimental disease is identical with that occurring spon-

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taneously under natural conditions, but that it ordinarily appears complicated with fowl-pox+diphtheria. Several investigators have attempted to discover the actual cause of contagious catarrh, but they have frequently taken cases of mixed infection as their starting point, so that none of the micro-organisms that has been found up to the present can with certainty be accepted as the cause of contagious catarrh.

The only bacillus that resembles a picture similar to that of contagious catarrh is the pasteurella. Nearly every investigator has, however, also obtained necrotic processes in the nasal channels, throat, and infra-orbital sinus. Van Heelsbergen informs us that with oval bacilli which he has cultivated from contagious catarrh, he has also produced nasal discharge, conjunctivitis, and œdema. The animals, however, died too rapidly from septicæmia, caused by these bacilli, for there to be time for the manifestations of contagious catarrh to develop completely. I examined several cases of contagious catarrh myself to ascertain if any of these oval bacilli were present, but failed to discover their existence. Out of four cases of contagious catarrh, de Zeeuw, at my instigation, once isolated an oval bacillus from the nasal mucus, which eventually killed the rabbit. He was, however, unable to produce contagious catarrh with this culture.

The cases of contagious catarrh I have referred to above are those, therefore, which could be transmitted in long series. Likewise, oval bacilli as well as other micro-organisms have been ætiologically examined. In a very short time after the manifestation of symptoms, the nasal mucus already contains such a variety of bacteria that it is extremely difficult to obtain a pure culture. In general there are a few species which grow so profusely that they undermine the nutritive media. Staphylococci are notably present, and at time bacillus proteus, streptococci, pseudo-diphtheria bacillus, and sometimes spirillum also. Several kinds of bacteria cultivated on agar and serum-agar in pure culture were incapable of producing, either alone or together, manifestations of contagious catarrh.

I performed a number of filtration experiments, filtering the nasal mucus of acute, subacute and chronic cases through various filters, among others also through the Seitz filter. I never succeeded in infecting a chicken with the filtrate of contagious catarrh. Knowing that the existence of a filtrable virus might be precluded, I tried to produce the disease with a mixed culture, twenty-four and forty-eight hours old, which I had obtained on agar and serum-agar plates by covering these with nasal mucus. This likewise proved to be a failure. More than once blood agar plates were also employed for cultures, but these were also very rapidly overspread by a rank growth of bacteria. After various attempts I succeeded at last in discovering some very fine colonies side by side with staphylococci and bacillus pseudo-diphtheria, colonies which in their growth strongly resembled the bacillus influenza of man. From this colony a pure culture of a bacillus was obtained, which, as far as my knowledge goes, has never been described in veterinary bacteriology. This bacillus in almost every respect is morphologically similar to the influenza bacillus of man. As regards its biology on an

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experimental nutritive media, this bacillus should undoubtedly be classed with the group of hæmoglobinophilic bacteria. No success was obtained when cultivating them on agar, serum-agar, in broth, or serum broth, but exclusively when nutritive media were used to which blood had been added. With pure cultures of this bacterium I have, up to the present, almost constantly been successful in producing contagious catarrh, whereby not only the nasal discharge, but also the typical œdema of the head were manifested. The nasal mucus of these culture-infected cases was again transmitted to fresh birds, while the same bacillus was once more discovered in this mucus. Infections with these cultures were successful both with regard to the fresh isolated culture as well as those cultures that had undergone twenty-five passages. As has already been mentioned, this bacillus is morphologically very similar to the influenza bacillus. It very much resembles a small coccobacillus. After a few culture passages it sometimes forms somewhat larger rods, or at other times shorter and longer threads. It is very pleomorphic, immobile, and Gram-negative.

The colony on blood agar and Levinthal agar is similar to that of bacillus influenzæ. The growth, however, is much less abundant. On Tildes agar I could not get a good culture. The best growth was seen on 25 per cent. horse blood agar, heated till 70° C. The colony is small, the size of a pin's head, colourless, and dewdrop-like. Its growth in blood broth is not very great. It attains its maximum growth after some forty-eight hours. I succeeded in producing infection both with blood broth and blood agar cultures. Even with a blood agar culture of some twelve days old, a chicken could be infected. According to my observations, the course of the experimental cases of the disease varies from six to eighteen days, after which period the symptoms disappear. One must, however, bear in mind that the birds were kept indoors, and under very favourable conditions. Several birds were likewise infected in the nostrils with staphylococci and pseudo-diphtheria bacilli from blood broth, which had grown on the original cultures with the hæmoglobinophilic bacteria. These chickens, however, and likewise the control birds which had been placed in separate cages in the same room, remained healthy.

I propose giving the bacillus with which contagious catarrh could be caused the name *Bacillus Hæmoglobinophilus Coryza Gallinarum*. Though this seems rather a long one, it appears to me to be very suitable, since it distinguishes it from many of the other bacilli which have already been described as being the cause of contagious catarrh. Further investigations are being carried out in order to discover what relation it bears, in conjunction with other bacteria, in infections which can be cultivated from the nasal mucus of catarrhal patients, and what course the experimental infection runs when the animals are kept, for instance, under more natural, but unfavourable conditions, such as cold, draughts, dampness, etc. Furthermore, the infection caused by contact, and under the same circumstances, will have to be controlled. Investigations are being undertaken in order to compare this bacillus with those hæmoglobinophilic bacteria already known in literature.

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Evidence will have to be obtained from further investigations as to whether in different cases of spontaneous catarrh this bacillus is manifested regularly. It will, however, be necessary to start from cases of pure contagious catarrh. The circumstances were not favourable for carrying out any such investigation at the time that the bacillus referred to above was discovered in consequence, of the disease being already on the wane in practice.

POULTRY PATHOLOGY AND THERAPEUTICS.

By HENRY GRAY, M.R.C.V.S.,
London.

THE poultry industry of this country is a great national asset which has not fully developed, as shown by the great imports of dead poultry and eggs from other countries. There is, therefore, great scope for the energy and talent of our people to supply the demands of the community for these products, and thus to prevent the country people thronging into large towns, and congesting further their already overcrowded population, and increasing unemployment.

It is true we have, during the past few years, increased the production many times, but as long as the great imports of these articles are still taking place, it is an indication that we are not satisfying the demand. What other countries can do, we ought to be able to cope with in these matters. Dumping should be prevented, to give our people a fair chance. It is much better for the community to increase labour by paying a little more for genuine home produced goods than to pay taxes towards keeping people on the dole just because we allow eggs and poultry from abroad, where they are produced by sweated labour, or by people who have a lower standard of living and a lower standard of taxation.

We ought to be able to produce all the poultry and eggs we require at a reasonable price, especially as there is so much land out of cultivation or under-cultivated, and corn-growing is at present so unremunerative. Why our farmers do not feed the poultry on home-grown corn, and thus save the corn merchants' or millers' profits, is a question for serious consideration. Nobody should be able to produce poultry and eggs so profitably as the general farmer, especially as he could grow all the food which they require when there was not sufficient by-products or waste found on the ground to maintain them in good health.

The greatest drawback to the industry are contagious diseases such as bacillary white diarrhœa, fowl-pox, typhoid, fowl plague, and, to a lesser degree, infectious neuro-lymphomatosis, contagious catarrh, and tuberculosis. Also to parasitic diseases like coccidiosis, trichomoniasis or "black-head," gapes, and some of the smaller helminthic infestations. Diseases like salpingitis, with impaction of the oviduct and peritonitis, leucæmia, gout, and paralysis, account for many deaths, but probably improper feeding accounts for more than all these complaints put

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together. The modern idea of feeding poultry is to see how well they can do on artificial concoctions of unnatural feeding stuffs, or to get away as far as possible from the simple, natural, or old system of nutrition. In-breeding or the improvement of breeds renders them more susceptible to disease, and the unnatural methods of feeding increase this susceptibility. Again, the artificial restraint a large number of highly bred fowls are put to on a very small area of ground increases the liability to transmission of infectious and parasitic maladies. It may be truly said that domestic poultry live under most unhygienic conditions, and, in consequence, losses in general are very great. It behoves the modern veterinarian to devote more attention to poultry than he has done hitherto in this branch of national wealth. There is no reason why some of the younger members, especially the women, of the profession should not concentrate on this. They should make themselves *au fait* with the breeds, anatomy, and physiology, dietetics, and breeding of the various domestic species. This they can do by attending such a place as the National Institute of Poultry Industry, Harper Adams Agricultural College, and learning all they can from those who have devoted themselves to these subjects. Then, after this, they should attend a special course of pathology and therapeutics, which should be given at all our veterinary schools, as is done in the Continental schools. The Ministry of Agriculture should be urged by the profession to grant travelling scholarships, so that our young men and women could spend some time studying the subject in the various veterinary schools or pathological institutes in other countries. It would be of great assistance to the small poultry farmer if county councils were to employ such poultry experts to advise on the spot the small animal owner how to overcome or prevent disease, or even on the management of small livestock. The rabbit owner, pigeon fancier, the cage-bird fancier, or bee keeper, should not be overlooked.

It is for the profession to create a demand of the community for the services of such veterinary specialists, whose work is of national importance. The Italians, Rivolta and Delprato, recognised its importance over fifty years ago when they wrote the first important work " L'Ornitoriatria o la Medicini ucelli Domestici e Semidomestici," Pisa, 1880. The two authorities distinguished on purely clinical grounds avian cholera from avian plague. Since then, Italy has devoted much labour in studying the nature of various epizoötics or enzoötics of different species of birds, even to proving that parrots were affected with a filtrable virus disease thought at the time to be transmitted from pheasants. Poultry pathology should also include the principal diseases of pheasants, partridges, grouse, and other avian species. The wild gallinaceous birds in this country are, from the experience of the writer, subject to all the infectious and parasitic diseases of domestic poultry. " Blackhead " is common in the various species of confined pheasant and quail, but it also occurs in the wild partridge that was bred and reared under natural conditions. Pheasants living up trees have tuberculosis, which runs a more chronic course than it does in domestic poultry. Coccidiosis

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occurs perhaps more frequently in the young pheasant than it does in the confined chicken. It seems to be more prevalent in dry, hot, than it does in wet, weather. It destroys a great number of partridge and grouse chicks. The various species of cage and aviary birds are frequently destroyed by coccidiosis, which in them is due to the genus *Isospora* and species *Lacazei*. The writer has witnessed it in the wild London sparrow found dying in the street, in the caged jay, strike, etc. Linnets, goldfinches, bullfinches, canaries, and other small birds are particularly prone to it in summer.

The gapeworm destroys a great number of young pheasants, partridges, grouse, and other birds. The writer has seen it in young nestling ornamental thrushes, birds of paradise, etc., in avaries. In young turdidæ it seems to destroy them just as they begin to get fairly covered with feathers while in the nest. No family of birds, whether wild, confined, semi-domesticated or domesticated, is immune to this parasite. It is sometimes encountered in adult pheasants and partridges, even in a sufficient number to occasion death by suffocation.

Fowl-pox or diphtheria also occurs in pheasants, partridges, wood-pigeons, and other wild birds. Avian plague is another disease to which they are subject, and destroyed in very large numbers. The young pheasant, as soon as it is turned adrift in some localities, manifests joint infection, attributed by Professor Gaiger to be due to a special micrococcus. It destroys a large number of birds, and may recur again the year following on the same preserve. The intestinal parasitic infestations, even if the parasites are of different species, are similar in the various species.

There are many other diseases of poultry that one sees the counterpart in wild species. But outside the confined birds, one rarely, if ever, encounters disease affecting the ovary or oviduct, or egg-binding. Pheasants suffer from an enzoötic form of blindness, sometimes showing pigmentation of the cornea, or at other times nothing to be detected amiss with the structures of the eyeball. Similar enzoötics to these are encountered in poultry on particular farms, or on more than one farm belonging to the same owner.

Country practitioners in whose districts there are large game preserves should know something of those diseases affecting game birds and furred animals, so as to be of assistance to the game preserver, as well as to earn fees and to gain for himself kudos for knowing more about other animals than the horse, cow, sheep or pig. When the writer entered the profession forty-six years ago the average practitioner felt himself belittled when asked to treat a dog for disease. As for a cat patient, it was an insult to him to be shown one. Since the displacement of the horse by motor traction, he was forced to the dogs and cats, which are being increasingly catered for by people outside the profession. This usurpation of our function is an indication of the intelligence of the general public, which seems more swayed by sentiment than by reason.

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BACTERIA OF THE ENTERIC GROUP IN POULTRY.

By C. A. McGAUGHEY.

From the Department of Bacteriology and Preventive Medicine, University of Manchester.

THE enteric bacteria most commonly found as the cause of disease in birds are *B. pullorum* and *B. gallinarum*, the conditions caused by these organisms being well known as " bacillary white diarrhœa of chicks," chronic " pullorum " disease of adult fowls, and " fowl typhoid." Effective control of these infections is based on the isolation and identification of the organism from infected birds, and on the detection of adult carriers by the agglutination test. If a suspension of *B. pullorum* is agglutinated, it is assumed that the bird may be a carrier of *B. pullorum* or *B. gallinarum*, as serological analysis has shown that the antigenic structure of these two organisms is identical. They possess a common " O " antigen. This antigen is also found in *B. typhosus*, *B. enteritidis* (Gaertner), and in the Dublin, Moscow, Dar-es-salaam and Tokyo types of Salmonella (White, 1926, 1929). It is evident, therefore, that the agglutination test as ordinarily carried out for the detection of carriers of *B. pullorum* may not be specific for this organism. The recognition of other enteric bacteria as a cause of disease in poultry is not only of bacteriological interest, but also of economic importance. Moreover, when species of bacteria which are concerned in " food poisoning " in man are found, the subject becomes still more important from the aspect of public health. It will be convenient to review the literature on this subject, and to record some personal experiences on infection of birds by enteric bacteria under three headings: (A) bacteria not usually pathogenic to man or animals; (B) bacteria of doubtful pathogenicity; (C) bacteria usually pathogenic to man and animals.

(A)

This group includes *B. coli* and similar organisms. These bacteria are normal inhabitants of the intestine of man and animals, and are also found in soil, water, milk, plants, etc. Table I shows the more important cultural characters of three of the commoner members of the *B. coli* group.

Pathogenicity.—Coliform bacteria have been incriminated by several authors as responsible for various acute and chronic infections in man and animals. Thus in man they have been isolated from the blood stream and organs in cases of cholecystitis (Moynihan, 1925, 1927); cystitis, pyelitis, etc. (Dudgeon, Wordley, and Bawtree, 1921, 1922, Dudgeon and Pulvertaft, 1927); while Jensen (1903), and Smith and Orcutt (1925) attribute diarrhœa in young calves to infection with *B. coli*. According to v. Heelsbergen (1929) many workers have found *B. coli* in the blood or organs of birds, *e.g.*, Ligières (1894) in hens, Sanfelice (1895) in pigeons, Florentini (1896) in swans and

geese, Martel (1897) in hens and turkeys, Spissu (1907) in ducks, Claussen (1907) in hens, Baudet (1913) in hens, v. Heelsbergen (1913) in ducks, v. Sande (1921) in hens, Zander (1922) in goslings, Palmer and Baker (1923) in hens. Most of these authors regarded the strains isolated as the cause of the various clinical conditions, and many of them showed that the cultures were pathogenic to birds, mice, rabbits, etc., when administered by various routes. Van Heelsbergen (1929), however, in a review of the recorded cases of "colibacillosis," is doubtful of the pathogenic significance of *B. coli*, and regards it as an agonal invader of the blood stream. On the other hand, it is probable that under certain conditions some strains of *B. coli* may develop invasive properties and become pathogenic. Dudgeon and his colleagues have shown that strains of *B. coli* from the fæces, or from the urine in cases of cystitis, may be divided into hæmolytic and non-hæmolytic types, and that the hæmolytic strains are more frequently found in certain cases of acute infection of the urinary tract. Similar studies of the strains of *B. coli* isolated from birds might reveal similar qualitative differences.

(B)

Under the heading of "Bacteria of Doubtful Pathogenicity," it is convenient to discuss the organism known as "Morgan No. 1," and closely related bacteria. The main characteristics of this group are: (1) the fermentation of dextrose, and, with the exception of Morgan No. 1, maltose and mannite; (2) no action or delayed action on lactose; (3) no action on salicin; (4) usually no action on saccharose. Motility, production of gas in carbohydrate media, production of indole, and action on litmus milk, vary with different strains.

These organisms may be regarded as somewhat intermediate between *B. coli* and *B. dysenteriæ*, and a convenient term for them is the paradysentery or dysenteroid group. In man, they have been found associated with dysenteric conditions (Morgan, 1905; Morgan and Ledingham, 1909; Lowenthal, 1912; Negre, Sergent, and Foley, 1916), and with infections of the urinary tract (Waaler, 1931). In animals *B. morgani* has been isolated by Scott (1926) from monkeys, by Wilson (1927) from mice, and by Lovell (1929) from several species of animals in the London Zoological Gardens. Table I shows the more important cultural characters of Morgan No. 1, and of several similar bacteria described by Morgan and Ledingham (1909), Lowenthal (1912), Negre, Sergent and Foley (1916), and Waaler (1931).

Personal Observations on the Presence of Paradysentery Bacteria in Fowls.

In a previous communication (McGaughey, 1930), an organism of the paradysentery group occurring in young chicks was described—Strain 1 in this communication. Five further strains have since been studied and compared with Strain 1.

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Origin of Strains.—Strain 1 was isolated in pure culture from the blood, organs, yolk residue, and intestines of young chicks which had presented the clinical features of bacillary white diarrhœa. Strain 2 was isolated in pure culture from the liver and other organs of young chicks from another flock similarly affected. Strain 3 was isolated from the liver and bone-marrow of an adult bird from a flock in which there had occurred an outbreak resembling fowl typhoid in its clinical and pathological features. *B. pullorum* was also isolated from the liver and bone marrow of this bird. Strain 4 was isolated from the intestines only of a fowl, one of several suspected to have been poisoned. No other organism of any significance was isolated. Strain 5 was isolated at The Wellcome Physiological Research Laboratories from the kidney and liver of an adult fowl. Several deaths had occurred in the flock; on post-mortem examination the kidneys were found to be swollen, and there was a white deposit on the heart wall. Strain 6 was isolated also at The Wellcome Physiological Research laboratories from the blood, liver, spleen, and bone marrow of three birds, none of which showed any abnormalities on post-mortem examination.

Morphology.—Short, stout rods with occasional longer forms arranged singly, non-sporing, non-capsulated, Gram-negative. All six strains were non-motile at 37° C. and at 25° C.

Colonies on agar, after twenty-four hours at 37° C. were round, opaque, greyish, slightly raised from 0.5 mm. to 2 mm. in diameter, with smooth surface and entire edge. After three days the colonies had increased in size up to 4 mm., the edges were crenated, and there was a faint fæcal odour. *Broth cultures* became turbid after eight hours at 37° C. After twenty-four hours there was a surface scum. After several days a heavy granular deposit was formed, and there was a faint fæcal odour.

Biochemical Reactions.—The more important reactions of the six strains, and also those of similar strains found in man and described by other observers, are shown in Table I. The following substances (1 per cent. solution in peptone water containing Andrade's indicator) were fermented in twenty-four hours with the production of acid and gas or acid only: glucose, maltose, mannitol, arabinose, levulose, galactose, glycerol. Strains 2, 3, 4, 5 and 6 also fermented lactose after 16 days at 37° C. with the production of acid only. Strain 1 did not ferment lactose even after thirty days at 37° C., and repeated passage through lactose solution had no effect. The following substances were not fermented: saccharose, salicin, ducitol, inositol, xylose, sorbitol, rhamnose, adonitol.

Litmus Milk.—The six strains were not all alike in their action on this medium. After twenty-four hours incubation at 37° C. all six cultures showed slight alkalinity at the surface of the medium. After forty-eight hours, Strain 1 became slightly acid, and then after three to four days slightly alkaline, remaining so for at least thirty days. Strain 2, litmus milk culture, after forty-eight hours, became more alkaline, and then after five days became decolourised; after twenty-five days it became acidified, but not clotted. Strains 3, 4, 5 and 6 also became

strongly alkaline, then decolourised after ten to fifteen days, and ultimately acidified and clotted in nineteen to twenty-one days. *Gelatine Stab.*—After twenty-four hours at 22° C. there was a fine filiform growth along the line of inoculation, but liquefaction did not occur even after incubation for twenty-one days. *Indole* was formed in peptone water cultures after three days. *Dextrose phosphate* cultures were methyl red positive, Voges-Proskauer negative. H_2S was not formed. *Nitrates* were reduced to nitrites. *Hæmolysin.*—All six strains showed hæmolysis on blood agar.

Agglutination Tests.—(*a*) A sample of serum from a three months old bird which had survived an outbreak (1) was tested for agglutination against the strain previously isolated from the chicks, and also against *B. pullorum*. There was no trace of agglutination against either suspension even with a 1 in 20 dilution of the serum. This bird was proved to be a carrier of the organism in the yolk residue (McGaughey, 1930).

(*b*) Agglutinating sera were prepared in rabbits against Strains 1 and 5, and tests were made with these two sera against all six suspensions, and against *B. pullorum*. The serum prepared against Strain 1 agglutinated the homologous suspension only as far as a dilution of 1 in 160, and showed no agglutination even in a dilution of 1 in 20 against the other strains. The serum prepared against Strain 5 agglutinated the homologous suspension up to a dilution of 1 in 5,120, but agglutinated the other strains only as far as a dilution of 1 in 80. Neither serum, even in a dilution of 1 in 20, showed any agglutination against *B. pullorum*.

Discussion.

Classification.—The six strains described were similar in morphology and, except in their action on lactose and litmus milk, in all cultural characters. Strain 1 resembled the organisms described as Group 14 (*a*) by Morgan and Ledingham (1909); the other strains were more like the organism described by Lowenthal (1912).

(*b*) *Serology.*—Only one bird, a survivor of outbreak (1) which proved to be a carrier, was examined serologically, but it had not developed agglutinins against the infecting organism. A similar failure to develop agglutinins has been observed in cases of human infection with bacteria of the paradysentery group (Gardner, 1929). Cross agglutination tests, using two sera against all six strains, revealed very little antigenic similarity between them, and none between them and *B. pullorum*. In this respect they resembled Morgan No. 1 (Morgan and Ledingham, 1909, Thjotta, 1920, Kligler, 1919, Dudgeon and others, 1919).

(*c*) *Pathogenicity.*—The circumstances in which Strains 1, 2, 5 and 6 were isolated suggest that the organisms described were ætiologically related to the clinical conditions, and were not merely agonal or post-mortem invaders. It is possible that these bacteria are normal inhabitants of the intestine, but they have not been encountered in cultures made from many young chicks and adult birds examined in this laboratory, nor is there any mention of similar organisms

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in communications by Rahner (1909), Menes and Rochlin (1929), and Emmel (1930) dealing with the intestinal flora of birds. Wilson (1927) suggests that *B. morgani* can give rise to infection in mice only in the presence of some predisposing factor which is probably associated with the dietary. It is possible that a predisposing factor is necessary in the case of infections in birds with the organisms described in this communication.

(C)

In the class of " enteric bacteria generally pathogenic to man or animals," it is necessary to discuss only one bacteriological group, *viz.*, the Salmonella bacteria. This large and important group includes: (1) species not pathogenic to man, but often found as the cause of various diseases in animals, *e.g.*, *B. pullorum* *B. gallinarum*, *B. abortus equi*, *B. abortus ovis*; (2) species which have been incriminated in food-poisoning in man, and some of which have been found to be the cause of disease in animals such as mice, rats, guinea-pigs, sheep, cattle pigs and birds. Thus *B. aertrycke* has been found in epizoötics in mice, guinea-pigs, and sheep; *B. enteritidis* (Gaertner) in rats and cattle, *B. suipestifer* in pigs.

In birds, infectious bacteria of this group have been reported in many species, but appear to be comparatively rare in gallinaceous birds. Nocard (1893) described an epizoötic disease in parrots from which he isolated an organism afterwards known as *B. psittacosis*, and recently proved to be identical with *B. aertrycke*. This organism was formerly regarded as the cause of psittacosis, but it has been definitely shown that the real cause is a filtrable virus. Beaudette (1926) described an epizoötic disease in canaries, in which he found *B. aertrycke*. According to v. Heelsbergen (1929), many authors have recorded infections by " paratyphoid " bacteria in these birds. This author also states that " paratyphoid " infections in pigeons are extremely common in Holland, Germany, and U.S.A., and that in a few cases the organism has been more exactly identified, *e.g.*, Beck and Meyer (1926) isolated *B. enteritidis* (Gaertner), and Beaudette (1926) isolated *B. aertrycke* from an outbreak in pigeons. An epizoötic disease of ducks known as " keel " in U.S.A., was described by Rettger and Scoville (1920), who isolated a Salmonella organism, and named it *B. anatum*. These authors suggested that recovered birds may become carriers and transmit the organism through the egg to their progeny. Some strains of *B. anatum* have been shown by Edwards and Rettger (1927) to be identical with *B. aertyrcke*. In an extensive outbreak of " keel " in ducklings in England, which was investigated by Gaiger and Davies (1930), an organism identical with, or closely related to, *B. anatum* (non-aertrycke type), was isolated from the affected birds. In a review of the literature on infections of birds with Salmonella bacteria, v. Heelsbergen (1930) states that Lerche and Spiegel described an epizoötic disease in ducks and geese from which *B. enteritidis* (Gaertner) was isolated. According to the same author, Pfeiler and Rehse (1913) isolated from diseased hens an organism of the *B. paratyphosus* β group, and Baudet isolated a similar organism from chicks with clinical signs of bacillary white diarrhœa. Baudet found that the hens from

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which the chicks had been bred were carriers, and that the infection had been transmitted through the egg. Lutje (1920) isolated *B. abortus equi* from hens affected with enteritis, and v. Heelsbergen (1930) found the same organism in chicks. Doyle (1927) described an outbreak in England in young chicks with the clinical signs of bacillary white diarrhœa from which he isolated *B. aertrycke*. The owner of the chicks informed him that many young ducks had died after showing clinical signs resembling those seen in the chicks, but none of the ducks was sent to the laboratory for bacteriological examination. Another case of *B. aertrycke* infection in chicks was recorded by Edwards (1929) in U.S.A. In this outbreak *B. anatum* (non-aertrycke type) was also isolated from the affected birds. No adult carriers could be detected by cultural examination or agglutination tests, and therefore Edwards came to the conclusion that the chicks had become infected after hatching.

Personal Observations on the Occurrence of Salmonella Bacteria in Poultry.

In the present communication five separate instances of infection of birds with Salmonella bacteria other than *B. pullorum* or *B. gallinarum* are recorded; three of them occurred in young chicks, one in an adult fowl, and one in a duck. Two of the outbreaks in young chicks, *viz.*, Nos. 1 and 2, were examined in this laboratory, the other cases were examined at The Wellcome Physiological Research Laboratories.

(1) This infection appeared in the form of an extensive outbreak in a flock of young chicks, about 50 per cent. of which died after showing clinical signs resembling those of "bacillary white diarrhœa." On post-mortem examination no striking abnormalities were noticed. *B. aertrycke* was isolated in pure culture from the liver and blood, and was identified by agglutination tests with specific absorbed serum prepared against a national type collection strain of *B. aertrycke* (Mutton). No samples of serum from the adult birds of the flock were available for agglutination tests. Six eggs were examined for the presence of the organism without success.

(2) This infection occurred in a flock of 300 chicks about seven days old. About 70 per cent. of them died within a few days after showing clinical signs resembling those of bacillary white diarrhœa. The chicks had been hatched together from three separate batches of eggs, and were all kept in the same brooder. On post-mortem examination no lesions were seen. A Salmonella organism, afterwards proved by cultural characters and agglutination tests to be identical with or closely related to *B. enteritis* (Gaertner), was isolated from the livers of the first three chicks examined. Three weeks later, three ailing chicks from the same flock were examined, but no significant organisms were found. About seven months later one of the few surviving pullets was examined. It was in very poor condition, and had not laid any eggs. On post-mortem examination masses of caseous material were found in the air sacs, and the ovary

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was small and shrunken. Cultures made from the caseous material, liver, gall-bladder, ovary, and intestinal contents were negative. A sample of serum from this bird was tested against suspensions of *B. enteritidis* (Gaertner) and *B. pullorum*, but no agglutination was detected.

(3) The details of this outbreak are not known, as only one chick was sent by the owner to The Wellcome Physiological Research Laboratories for routine examination. No noteworthy abnormalities in the organs were found, but cultures from the heart blood, liver, and lungs yielded a growth of an organism which proved to be an unusual member of the Salmonella group. Although it has been frequently subcultured during the last twelve months, it has remained non-motile both at 37° C. and 25° C. Colonies on agar after twenty-four hours at 37° C. were 2–3 mm. in diameter, round, with entire edge and smooth surface, later increasing up to 4 mm., and the edge becoming scalloped. In its action on carbohydrates, the organism resembled *B. aertrycke*, *B. paratyphosus*, *Hirschfeld L*, *Derby*, *Reading* and *Morbificans bovis* (*see* Table II). The results of agglutination tests indicated that it was related to *B. aertrycke*, *B. paratyphosus* β, and other Salmonella bacteria containing identical " O " antigens.

(4) In this outbreak a number of adult birds had died suddenly, and several others were ill. From the liver and spleen of one bird examined at The Wellcome Physiological Research Laboratories a Salmonella organism was isolated. By cultural characters and agglutination tests with specific serum it was identified as *B. aertrycke*. Six more birds were examined; from two of them *B. pullorum* only was isolated, but cultures from the other four birds were negative. Samples of serum from sixty birds in this flock were tested against *B. pullorum* and *B. aertrycke*. Sixteen of them agglutinated *B. pullorum* only, and one agglutinated with *B. pullorum* and *B. aertrycke*. The remaining forty-three samples were negative against both organisms.

(5) This infection occurred in ducks. The history was that a number of ducks had died in the course of several months. One bird was sent to The Wellcome Physiological Research Laboratories for examination. On post-mortem examination the only lesion seen was an abnormal appearance of the ovary, which resembled the misshapen ovaries seen in hens with chronic *B. pullorum* infection. A Salmonella organism was isolated from the liver and ovary, and was found to be identical with or closely related to *B. enteritidis* (Gaertner) by cultural and agglutination tests. The more important fermentation reactions of the commoner members of the Salmonella group, and also those of the strain isolated in outbreak (3), are shown in Table II.

Discussion.

(*a*) Incidence of Infections in Poultry with Salmonella Bacteria other than B. Pullorum and B. Gallinarum.

As v. Heelsbergen (1929) has already pointed out, infections with bacteria of the " food-poisoning " group have been more frequently recorded in ducks and geese than in gallinaceous birds. It has been suggested by Lecoq, in a

monograph on food poisoning in man attributed to ingestion of contaminated eggs, that the reason for the greater frequency of such infections in ducks is their constant association with ponds and ditches which may be contaminated by human excreta. This author was of the opinion that ducks could be infected by passage of the organisms up the oviduct. Rettger and Scoville (1920), however, suggested that ducks which had survived an outbreak of " keel," and had become carriers of the organism in the ovary, might transmit the infection through the egg. The observations of Baudet, previously mentioned, suggest that a similar method of transmission may occur in hens.

(*b*) Diagnosis of Infections with Organisms other than B. Pullorum and B. Gallinarum.

The other Salmonella bacteria are easily isolated on the same media which are commonly used for the isolation of *B. pullorum* and *B. gallinarum*. The colonies are generally larger and more opaque than those of *B. pullorum*. Their characteristic motility and production of gas in carbohydrate media distinguish them at once from *B. gallinarum*, and the production of acid and gas in maltose and dulcitol from *B. pullorum*.

Complete identification may be established by agglutination tests with specific sera. Not much work has been carried out on the detection of carriers by the agglutination test, but in view of the antigenic relationship of *B. enteritidis* (Gaertner) to *B. pullorum* and *B. gallinarum*, it is probable that the serum from carriers of *B. enteritidis* (Gaertner) would agglutinate *B. pullorum* and *B. gallinarum*.

(*c*) Relation to Food-Poisoning in Man.

Little information on this subject is available. According to Scott (1930), cases of food poisoning in France caused by ingestion of whipped cream were proved by Lecoq (1906) to be caused by bacterial contamination of the egg albumen used in the mixture. Apparently both hens' eggs and ducks' eggs were incriminated by this author. Scott also referred to a case of food poisoning in Yorkshire in 1913, which was investigated by Spencer Low and attributed by him to the use of infected egg albumen in cream buns. Scott (1930) himself described seven more or less extensive outbreaks of food poisoning caused by *B. aertrycke* in which suspicion could be attached only to ducks' eggs as the source of infection. He also suggested that many solitary cases of food poisoning may be due to the eating of an infected egg. With regard to food poisoning from infected poultry, still less is known. v. Heelsbergen (1929) states that Hubner (1910) described eleven cases of food poisoning attributed to the ingestion of infected poultry, but it was not proved whether the birds had been infected before or after death.

Summary.

(1) Infections of poultry with bacteria of the enteric group other than *B. pullorum* and *B. gallinarum* are discussed. (2) Six strains of bacteria, isolated from chicks and adult fowls in six separate infections and resembling the organisms

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TABLE I.

REACTIONS OF ORGANISMS OF B. COLI AND PARADYSENTERY GROUPS.

Species or Description of Organism	Motility	Lactose	Glucose	Mannitol	Sucrose	Salicin	Dulcite	Litmus Milk	Indole
B. coli commune	+	AG	AG	AG	O	AG	AG	AC	+
B. coli communior ...	+	AG	AG	AG	AG	O	AG	AC	+
B. acidi lactici	—	AG	AG	AG	O	O	O	AC	+
Morgan No. 1	+	O	AslG	O	O	O	O	O or A—Alk	+
Morgan and Ledingham's Group 14 ...	+	O	AG	AG	O	?	O	A—Alk	+
Paradysentery bacillus (Lowenthal)	—	O—A	AG	A	O	—	O	A—Alk A—C	+
Paradysentery bacillus (Negre, Sergent and Foley)	+	AG	AG	AG	?	—	?	AC	?
Waaler's strain (1931) ...	+	O	AG	AG	AG	—	O	?	+
Strain (1) chick	—	O	AG	AG	O	O	O	sl. Alk	+
Strain (2) chick	—	A(late)	AG	AG	O	O	O	Alk-decol; A (late)	+
Strain (3) fowl	—	A(late)	AG	AG	O	O	O	Alk-decol; AC (late)	+
Strain (4) fowl	—	A(late)	AG	AG	O	O	O	Alk-decol; AC (late)	+
Strain (5) fowl	—	A(late)	AG	AG	O	O	O	Alk-decol; AC (late)	+
Strain (6) fowl	—	A(late)	AG	AG	O	O	O	Alk-decol; AC (late)	+

AG=Acid and gas.
A=Acid.
AC=Acid and clot.
Alk=Alkaline
?=not recorded.
A—Alk=Acid changing to Alkaline.
O=No change.
+=Positive reaction.
—=Negative reaction.

TABLE II.

TYPICAL REACTIONS OF SOME SALMONELLA ORGANISMS.

Species.	Motility	Lactose	Glucose	Maltose	Mannite	Sucrose	Salicin	Dulcite	Inosite	Litmus Milk	Indole
Pullorum	—	—	A or AG	—	A or AG	—	—	—	—	A	—
Gallinarum	—	—	A	A	A	—	—	A	—	A—Alk	—
Enteritidis (Gaertner) ...	+	—	AG	AG	AG	—	—	AG	—	Alk	—
Abortus equi ...	+	—	AG	AG	AG	—	—	AG	—	Alk	—
Paratyphosis β ...	+	—	AG	AG	AG	—	—	AG	AG	Alk	—
Aertrycke	+	—	AG	AG	AG	—	—	AG	AG	Alk	—
Anatum	+	—	AG	AG	AG	—	—	AG	AG	Alk	—
Strain from chick in case (3) ...	—	—	AG	AG	AG	—	—	AG	AG	Alk	—

A=Acid.
Alk=Alkaline.
+=Positive reaction.
AG=Acid and gas.
A—Alk=Acid changing to alkaline.
—=Negative reaction.

isolated from man by Morgan (1907), Morgan and Ledingham (1909), are described. (3) Two of the six strains were isolated in pure culture from the blood, organs, and intestines of young chicks with clinical signs of bacillary white diarrhœa; the remaining four strains were isolated from the blood, organs, or intestines of adult fowls. (4) Agglutination tests have shown that there was little antigenic relationship between the six strains. (5) The pathogenicity of the six strains is discussed, and it is suggested that in four of the cases described the organism isolated was the cause of disease. (6) Five instances of infection with bacteria of the Salmonella group are described. (7) In two instances, one an outbreak in young chicks showing clinical signs of " bacillary white diarrhœa," *viz.*, Case (2), and the other, a chronic disease in ducks, *viz.*, Case (5), an organism which by cultural and serological tests seems to be identical with, or at least closely related to, *B. enteriditis* (Gaertner), was isolated. In one case, *viz.*, Case (3), a non-motile Salmonella organism, which by cultural and serological tests seems to be related to *B. aertrycke*, was isolated. Further serological tests are necessary for the complete identification of the organisms isolated in Cases (2), (3) and (5). (8) Infections of birds with enteric bacteria other than *B. pullorum* and *B. gallinarum* are of interest to veterinarians for several reasons, *viz.*: (i) They cannot be differentiated clinically from " Pullorum " disease or fowl typhoid, both of which diseases are of great economic importance. (ii) The effective control of these diseases depends on early diagnosis. (iii) The possibility of spread to, or infection from, other animals must be considered. (iv) The origin of infection in certain cases of food poisoning has been traced to infected birds or eggs.

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FOWL CHOLERA IN ENGLAND.

By G. H. WARRACK, B.Sc., and T. DALLING, M.R.C.V.S.
Langley Court, Beckenham, Kent.

DURING the past year four outbreaks of a disease which we believe to be fowl cholera have come to our notice. In each case we have isolated a bipolar staining micro-organism which we believe by its various characters and reactions to be *Pasturella avium* (*B. avisepticus*).

Outbreak I. (Sub-Acute Fowl Cholera).

Two adult hens were received from Mr. Tr. in December, 1930. We noted an enteritis, but were unable to isolate any pathogenic micro-organism from the tissues examined. In February, 1931, another bird was received, with the information that losses were occurring at the rate of about six per week, and several birds were now ill. The disease had been confined to one house originally, but recently had spread to every house. In all, some 60 out of 500 birds had died. The only symptom shown during life was a mopy condition of the birds, which simply " stood about " for a few days and then died. Several, but not all, showed diarrhœa before death. In the badly affected house the birds were still laying at the rate of 60 per cent. Some of the dead birds appeared to have developed " colds." Before death the birds had refused all food.

From the heart blood, liver, spleen and lungs of this bird, a bipolar staining organism was isolated. It was identified as *P. avium* by its " sugar " reactions and its pathogenicity in other animals : minute amounts of culture killed pigeons, mice and rabbits, from whose blood the bipolar staining organism was again isolated. Our findings were conveyed to the owner, and certain measures recommended. We heard no more from him, but from an indirect source we learned that the losses continued. Over a year after the first losses occurred, we received a live cockerel from Mr. Tr. with the information that this bird was one of four recently purchased, and had become ill three days after its arrival. When received at the laboratory it was found *in extremis*, and was killed forthwith. From its spleen a pure culture of *P. avium* was readily isolated. The points

in connection with this outbreak are its sub-acute nature (in typical outbreaks the losses are 90–95 per cent.), and its persistence, for it seems evident that the disease still exists on the farm.

Outbreak II. (Sub-Acute Fowl Cholera).

In March, 1931, two hens were received from Mr. Tu., with the history that they were in a lot isolated because twelve deaths had occurred during the past two months. This lot of birds had been bought locally, and had been penned by themselves, with no direct contact with the remainder of his stock. We isolated *P. avium* from the heart blood, liver, and bone marrow of the two hens, as well as from similar tissues from a further two hens received several days later. Since March we have received several dead birds from Mr. Tu., taken from various pens on the farm, but in no case have we been able to diagnose fowl cholera. It would appear that this outbreak was confined to one group of birds, and that the disposal of these birds removed the source of infection, and prevented further spread.

Outbreak III. (Œdema of the Wattles).

Mrs. H. sent us a dead cockerel in October, with the history that two out of thirty bought recently had died after showing symptoms of a slight "cold," followed by a loss in condition. The colds were observed in others in the group. The lungs of this bird appeared very much inflamed, and from its various tissues *P. avium* was isolated. Two live cockerels from the same group were received a fortnight later. In both the wattles were swollen and wrinkled. The birds are still alive. We attempted to culture the contents of the swollen wattles, but with negative results. From our experiences in Outbreak IV. (*see* below) we are of opinion that the wattle condition is that described by Ward and Gallacher (1922), and by Grasovsky (1931).

Outbreak IV.

Mr. R. sent us six live cockerels whose wattles were much swollen, with the information that he had several more affected. We found the wattles in various stages of tumefaction (two were remarkably swollen), and were very similar to those described by the above authors. Only one of the six birds appeared ill. It was killed, and swabs from its trachea, as well as material from the wattles, yielded cultures of *P. avium*. Further, a sample of the debris contained in the wattle of one of the birds taken during life also yielded typical cultures of *P. avium*.

Conclusions.

(1) Fowl cholera (caused by *Pasturella avium*) exists in England as a sub-acute disease, and causes death among fowls. (We have no record here of its occurring as an acute epidemic.) (2) Infection may persist among infected stock for at least one year, as evidenced by the infection of newly introduced stock into affected premises. There is some evidence that the disease is more acute in newly introduced birds. (The cockerel in Outbreak II. became ill three days after its arrival.) (3) In some cases the so-called colds among fowls may

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be sub-acute fowl cholera (throat swabs may yield cultures of *P. avium*). (4) Œdema of the wattles in cockerels may be caused by infection with *P. avium*, which may be isolated in pure culture from the debris contained within the swollen wattles.

(We feel that fowl cholera may be more common in England than is generally thought.)

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SO-CALLED "FOWL PARALYSIS."

Also called Neuritis in Chickens, Range Paralysis, Neuro-Lymphomatosis Gallinarum.

A DISCUSSION ON THE VARIOUS THEORIES AS TO CAUSATION, WITH SPECIAL REFERENCE TO FIELD OBSERVATIONS AND LABORATORY TRANSMISSION EXPERIMENTS.

By G. H. WARRACK, B.Sc., and T. DALLING, M.R.C.V.S.

Langley Court, Beckenham, Kent.

Introduction.

WHILE paralysis occurs in fowls as a symptom or result of a variety of conditions, it is now generally accepted that the condition commonly referred to as "fowl paralysis" or "neuro-lymphomatosis gallinarum" can be differentiated from those presenting somewhat similar features. The condition under discussion is that already described by Doyle (1926), Pappenheimer and his colleagues (1929), and others in America, and Galloway (1929); McGaughey and Downie (1930), and Bayon (1931) in this country. Galloway (1929), who was the first to refer to its existence in this country, describes the histological findings in the affected tissue, and also claims to have succeeded in transmitting the disease to fresh birds.

Since we first encountered this disease early in 1930, we have met it many times. Some 76 outbreaks have come to our notice through routine post-mortem examination work, and we have in our possession detailed records of 40, while we have made a special study of 10. Several hundred post-mortem examinations have been made, many sections have been cut and examined, and we have carried out experiments on transmission under laboratory and field conditions.

Diagnosis.

We do not propose to discuss the symptoms and general characters of the disease here. These have already been described by Doyle (1926 and 1927), McGaughey (1930), Bayon (1931), and several others, including ourselves (1931). We would, however, briefly describe what constitutes, in our opinion, the three

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characteristic features of the disease, and on which we base our diagnosis of naturally occurring and artificially produced fowl paralysis; clinical symptoms, especially concerning the entire flock in which the outbreak occurs; post-mortem findings; and some histological appearances. Naturally, all outbreaks do not present exactly the same clinical features. Lameness in one or both legs is fairly characteristic, and was present in 78 per cent. of the outbreaks with which we dealt. Lame birds, as a rule, have a perfectly healthy appearance, but the lameness is progressive, and ultimately in days or weeks a general paralysis is found accompanied by rapid and marked emaciation. The general healthy appearance of the affected birds usually persists, and if hand-fed they may live for weeks. (We have kept paralysed birds alive at the laboratory for three months.) Some lame and paralysed birds may show evidence of digestive disturbances, *e.g.*, swollen, flaccid crops, diarrhœa, etc., and in some few birds such symptoms may be the first observed. In our opinion, in such cases lesions will usually be found involving the digestive tract or its control. The drooping of one or both wings will usually be observed in some birds. The drooping is characteristic in that if the wing be placed in a normal position it will quickly return to its original drooping state. Drooping wings seldom if ever become normal: a general paralysis usually supervenes.

Iritis is a fairly common symptom. While in some cases recovery takes place, as a rule the condition rapidly becomes chronic, when the name "white or opaque eye" is applied. It is held by some that the eye condition is in itself diagnostic of the existence of fowl paralysis in a flock. Paralytic symptoms affecting the head and neck may be seen in some birds, causing a twisting of the head, or an inability to raise the beak from the ground. Some writers record increased excitability. We are not convinced that this occurs in true uncomplicated fowl paralysis.

The chief post-mortem diagnostic lesions are the swelling of peripheral nerves and nerve ganglia, especially those of the limbs, and though the condition is usually unilateral, we have many records of its occurrence bilaterally. We also attach considerable importance to the occurrence of tumours—usually of a fairly soft nature—involving any of the internal organs or tissues or the skin. We have records of such tumours occurring in the ovary, kidney, mesentery, liver, pancreas, gizzard, and intestinal wall. In many instances tumours have been found in birds which also showed swollen nerves. Many of these tumours are composed of the same type of cells as found in the enlarged nerves, etc. Some authors attach much importance to the condition of the duodenum, and describe catarrhal conditions, actual duodenitis, and sloughing of the mucous membrane. In 30.3 per cent. only of the cases examined did we find any abormnal condition of the duodenum.

In all outbreaks we confirm our diagnosis by histological examination of suitable material, *e.g.*, brain (cortex), peripheral nerves, ganglia, etc., as well as tumours. Evidence of accumulations of cells of the lymphocyte, mononuclear, and plasma

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type is accepted as diagnostic of fowl paralysis. As a rule, there is little difficulty experienced in demonstrating collections of such cells. In the brain cortex the numbers of such cells may on occasion be comparatively few. Some difficulty in diagnosing may be experienced in the absence of the typical "cuffing." In cases of doubt, however, it is usually a fairly easy matter to procure suitable material from other affected birds in the flock. Collections of such cells may also be found in tissues such as the liver, kidney, etc.

Causation.

Some observers attach considerable importance to the association of intestinal parasites with fowl paralysis, and some even hold that such parasites constitute the sole cause. Lee (1931) found extensive catarrhal enteritis present in practically every case of fowl paralysis, and he thinks that toxins elaborated in the intestine are allowed to pass through the damaged intestinal wall. He also believed that the most common cause of the enteritis is coccidia. In the few cases which show no parasites, he suggests that the catarrhal enteritis is due to dietary origin. Niemann (1929) found that in at least 50 per cent. of the fowl paralysis flocks he examined, coccidiosis was present, and in many tapeworms were also found. He thinks it possible that there is no single ætiological factor, and that the controlling of coccidia and intestinal parasites will reduce the incidence of fowl paralysis. He adds, however, "the possibility of a filtrable virus being the ætiological factor is not to be altogether ignored, especially in the absence of definite information relative to other factors." Stafseth (1931) showed by post-mortem work that leg weakness or paralysis is associated largely with duodenal coccidiosis, and gives details of some histological work done in connection with the nervous tissue of birds affected with duodenal coccidiosis. He says that "enlargement of nerves was observed quite often, undifferentiated mesenchymal cell infiltration was found in the brain, spinal cord, and peripheral nerves, sometimes even thickening of these nerves was noticeable."

Some observers, on the other hand, negative the idea that intestinal parasites and fowl paralysis have any inter-relationship whatever. May, Tittsler and Goodner (1925) showed that there was "no essential difference in the number of worms present in paralytic and non-paralytic birds"; that "coccidia are frequently present in paralysed birds, but not in all," and, furthermore, "birds may die from coccidiosis without exhibiting any symptom of fowl paralysis." Beaudette and Hudson (1931) consider that the disease constitutes a clinical entity, and has one primary cause. They say that "intestinal parasites and coccidia have been incriminated, and though it is not denied that these may occasionally cause paralytic symptoms, they are not primarily concerned with the disease in question." They add that "typical fowl paralysis occurs in spite of the absence of coccidia and other parasites." Bayon (1931) states that "the observations of neuro-lymphomatosis in the field seem to indicate that though several different parasitic infestations, but more particularly Davainea proglottina and coccidia, may initiate paralysis, yet the acute and chronic forms of disease,

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neuro-lymphomatosis gallinarum, may develop without the presence of such parasites." He further states that nutritional factors may also play a part in the disease, and adds: "but, however important, these also do not explain its appearance in single pullets of a batch, all of which are fed in the same manner. Such questions cannot be answered otherwise than by experiments . . ." Lately, Blount (1931) put forward the suggestion that "one factor in the causation of certain avian diseases, among which may be included so called fowl paralysis, is the feeding of grit of improper constitution. Devitalisation of the intestines occurs, and toxins are absorbed into the general system."

In the course of our investigations we have paid much attention to the question of the presence of parasites in the intestine, the state of the duodenum, and the feeding methods adopted in the various affected flocks. We found in the birds we examined from thirty-three infected flocks (and on many occasions several birds from a flock were examined), the following was the result: Coccidia in the cæca in 23, or 69 per cent.; coccidia in the duodenum in 10, or 33.3 per cent.; cæcal worms in 5 or 15.1 per cent.; tapeworms in 5, or 15.1 per cent.; round worms of various species in 4, or 12.1 per cent. In 9, or 27.2 per cent., no parasites of any recognised variety could be demonstrated. Further, in some of the outbreaks it was necessary to examine several birds before one could be found harbouring any intestinal parasites. (In our search for coccidia in the duodenum, all the usual methods, including the histological examination of sections, have on occasion been carried out, with negative results as already indicated.) Our records show that the methods of rearing, feeding, and general managements of different flocks in which fowl paralysis exists may be entirely different; thus the disease has been demonstrated in flocks where birds have been reared intensively all their lives, in flocks on free range at all times, and in fowls reared intensively for the early part of their lives, and then allowed free range. It is no uncommon occurrence to find that birds from a special source (*see* Appendix I. (*d*) of birds bred from an individual parent (*see* Appendix II.) are the only birds to develop symptoms of the disease, while living under exactly the same conditions, and in intimate relationship with all the other birds hatched and reared in the same machines, and in the same houses. From the above observations we feel we cannot agree that coccidia or any other intestinal parasites are primarily concerned in the causation of fowl paralysis.

Transmission.

Early in our work we attempted transmission experiments. There are several records of the disease having been transmitted under laboratory conditions.

Siefried (1929) injected year-old hens intracranially with brain, cord, and lumbar and sacral swellings emulsified in saline, and fed others with the same emulsions. He claims to have reproduced the disease in five out of five hens fed, and three out of the fifty-one injected. The incubation period was four to twelve weeks. Later he repeated his former successful feeding transmission experiments, using chicks seven weeks old, and concludes that feeding is the most constant

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method of transmission, and that contact does not reproduce the disease. He adds that : " the facts all point to a specific causal agent with a high glycerol resistance, probably a filtrable virus." Pappenheimer and his colleagues (1929) succeeded in transmitting the disease by subdural inoculation into young chicks of nerve tissues from affected cases. They base their diagnosis of infection having taken place on the results of microscopic examination of brain and nerve sections, and found that the typical cells associated with fowl paralysis (lymphoid, plasma, and mononuclear cells), though present in some control birds to a very slight degree, developed to a marked extent in many of the inoculated birds when examined within two months of treatment. (Taking only the unmistakable and characteristic positive cases, they found in 89 inoculated birds 24 positive, *i.e.*, 26 per cent., while in 104 birds kept under comparable conditions, only seven, or less than 8 per cent.) They state that they feel justified in drawing the conclusion that the inoculation of the material from paralysed fowls definitely favoured the subsequent development of the disease, and " while the evidence, epidemiological and experimental, points to an infective agent, we are totally ignorant as to its nature, and as to the manner in which the disease is conveyed under natural conditions." Galloway (1929) states that he transmitted the disease by intracerebral inoculation of emulsions of the nervous system of diseased birds.

We also have succeeded in transmitting the disease. Our first attempts were made on adult fowls (over one year old), and our results were sufficiently encouraging to warrant further experiments in which younger birds (2–2½ months old) were used. Our diagnosis of the artificially-produced disease has been made from clinical features, post-mortem lesions, and histological work. We have not relied entirely, as did Pappenheimer and his colleagues, on histological evidence. We feel that had we done so, we would have been able to record a considerably greater number of positive cases. We, like Pappenheimer, found in the brains of a small percentage of so-called normal birds some slight evidence of the presence of the typical fowl paralysis type of cells. It is true that they were only exceptionally found and in small numbers ; still, because of this complication, we have disregarded as positive cases treated birds which have shown histological without typical clinical or post-mortem evidence, and have regarded as positives only such treated birds as presented unmistakable clinical and post-mortem features. The following is a record of some of our transmission experiments :

Experiment I.

Preliminary—adult birds used. Material used was from four chronically affected birds, and was shown to be sterile.

(*a*) *Spleen* (emulsion in saline).—1 c.c. injected into two birds intravenously (Nos. 1 and 2), and into one subcutaneously (No. 3). No. 1 died three months later without showing any definite symptoms or lesions. Its spleen injected into two birds produced no ill effects within three months. Nos. 2 and 3 showed lameness, but recovered.

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(*b*) *Lungs* (emulsion in saline).—One c.c injected into three birds intravenously (Nos. 4, 5 and 6), and into one subcutaneously (No. 7). Nos. 4 and 5 died within a few minutes after injection. No. 6 showed lameness and recovered. No. 5 (" white spiral " in our records) behaved normally till three months after injection, when it showed lameness and gradually developed into a typical case of fowl paralysis. (*See* Appendix III.)

(*c*) *Brain* (emulsion in saline).—0.1 c.c. injected intra-cranially into nine fowls (Nos. 7, 8, 9, 10, 11, 12, 13, 14 and 15). Nos. 7, 8 and 12 died within one week. The brain of No. 12 was passaged intra-cranially in another bird without any positive results. Nos. 9, 10 and 11 all limped for some time, but recovered and remained well. Nos. 13, 14 and 15 showed no ill effects.

The result of this preliminary experiment was that a typical case of clinical fowl paralysis developed in a bird three months after being injected intravenously with a lung emulsion from four chronic naturally occurring cases.

It was decided at this stage to attempt " passage " experiments, using the tissues of bird No. 5. Spleen and lungs emulsions were made and injected as under :

(*a*) *Spleen.*—Injected intravenously into birds 16 and 17. No. 16 died in four weeks, and No. 17 in four months without showing typical symptoms.

(*b*) *Lungs.*—Injected subcutaneously into birds Nos. 18 and 19. No. 18 has never shown any ill effects. No. 19 died in four weeks after having exhibited paralytic symptoms involving the head and neck.

(*c*) *Spleen and Lung* (*Mixture*).—Injected intramuscularly into birds Nos. 20 and 21. No. 20 showed paralytic symptoms involving the head and neck in two months, and died. No. 21 died six months later after showing typical clinical symptoms and post-mortem lesions. (*See* Appendix III.)

The result of this passage experiment is that at least one, and probably three, of the six birds injected developed symptoms and lesions characteristic of fowl paralysis. (Six months elapsed between the injection and the evidence of infection.)

Experiment II.

This experiment embraces the use of 96 birds, 2–2½ months old, 71 of which were treated, and 25 were kept as controls. The control birds were housed and fed in a manner exactly similar to those treated, and the treated and untreated were attended by the same person. All birds were from the same hatches. The experiment can be divided as follows :

(A) *Material from chronically infected birds :* (*a*) Spleen—injected (i) intravenously ; (ii) intramuscularly. (*b*) Lungs injected intramuscularly. (*c*) Brain —injected (i) intra-cranially ; (ii) subcutaneously ; (*d*) Peripheral nerves—injected (i) intra-cranially ; (ii) subcutaneously ; (iii) fed.

(B) *Material from early cases :* (*a*) Spleen—(i) injected intravenously ; (ii) fed. (*b*) Lungs—(i) injected subcutaneously ; (ii) fed. (*c*) Brain—injected (i)

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intra-cranially ; (ii) subcutaneously ; (iii) fed. (*d*) Peripheral nerves—injected (i) intra-cranially ; (ii) subcutaneously ; (iii) fed.

(A) *Material from Chronically Affected Birds.*—These birds had been under our observation for some weeks, and were regarded as typical cases showing chronic symptoms and lesions. Emulsions of the various tissues used were made in saline, and the quantities were : 1 c.c. injected intravenously, subcutaneously, or intramuscularly ; 0.1 c.c. injected intra-cranially, and about 5 c.c. fed.

(*a*) *Spleen.* (i) *Injected Intravenously.*—Birds 22, 23, 24 and 25 used. Nos. 22 and 23 remained normal. No. 23 showed definite symptoms and lesions of infection in two months and nine days (*see* Appendix III.). No. 24 showed evidence of head and neck paralysis in ten weeks and was killed. (ii) *Injected Intramuscularly.*—Birds 25, 26, 27 and 28 used. No. 25 was lost. No. 26 developed definite symptoms and lesions of fowl paralysis in eight weeks (*see* Appendix III.). No. 27 died after three months—no record of any definite symptoms or lesions. No. 28 showed some indefinite symptoms in three months, and died fourteen days later.

(*b*) *Lungs. Injected Intramuscularly.*—Birds 29, 30, 31, 32 and 33 used. No. 29 developed head and neck symptoms in eight weeks and was killed. No. 30 died in nine weeks ; no record of symptoms or lesions. No. 31 was killed accidentally nine days after injection. No. 32 died in five months ; we do not know the cause. No. 33 was lost.

(*c*) *Brain.* (i) *Injected Intra-cranially.*—Birds 34, 35, 36, 37 and 38 used. Nos. 34–37 died within forty-eight hours. No. 38 died in five and a-half months ; no typical symptoms or lesions noted. (ii) *Injected Subcutaneously.*—Birds 39, 40 and 41 used. Nos. 39 and 40 died during seven days. No. 41 died in ten weeks ; no diagnostic symptoms or lesions found.

(*d*) *Peripheral Nerves.* (i) *Injected Intra-cranially.*—Birds 42, 43, 44 and 45 used. All died within forty-eight hours. (ii) *Injected Subcutaneously.*—Birds 46, 47 and 48 used. No. 46 died in six days. No. 47 died in ten weeks ; no symptoms or lesions of a diagnostic character found. No. 48 showed no ill effects. (iii) *Fed.*—Birds 49 and 50 used. No. 49 died in one week. No. 50 died in six weeks without showing any diagnostic symptoms of post-mortem lesions.

(B) *Material from Early Cases, i.e.,* birds which had shown definite symptoms for a few days only. Emulsions made and amounts injected as above.

(*a*) *Spleen.* (i) *Injected Intravenously.*—Birds 51, 52, 53 and 54 used. No 51 in nine weeks developed typical clinical symptoms, and was positive on post-mortem examination (*see* Appendix III.). No. 52 died in fourteen days. No. 53 died in ten weeks ; nothing characteristic on clinical or post-mortem seen. No. 54 has never shown anything abnormal. (ii) *Fed.*—Birds 55, 56, 57 used. No. 55 showed definite evidence of head and neck paralysis in nine weeks. No. 56 was lost. No. 57 died in five months ; our notes do not record any special symptoms or lesions.

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(*b*) *Lungs.* (i) *Injected Subcutaneously.*—Birds 58, 59, 60 and 61 used. In thirteen weeks No. 58 developed typical symptoms, and was a positive case on post-mortem (*see* Appendix III.). No. 59 died in two months. No. 60 in two and a-quarter months; we do not know the cause. No. 61 still survives. (ii) *Fed.*—Birds 62, 63 and 64 used. The birds died at intervals of one week to three months without showing any definite clinical symptoms.

(*c*) *Brain.* (i) *Injected Intra-cranially.*—Birds 65, 66, 67, 68, 69, 70, 71 and 72 used. Nos. 65–67 died within seven days. No. 68 died in ten weeks after showing "head" symptoms. Nos. 69 and 70 died in five months; nothing of a diagnostic nature recorded. No. 71 died in five and a-half months without showing any definite symptoms. No. 72 has survived. (ii) *Injected Subcutaneously.*—Birds 73, 74, 75 and 76 used. No. 73 died in three weeks. No. 74 died in two months. No. 75 died in thirteen weeks. None of these birds showed any characteristic symptom or post-mortem lesion. No. 76 is still alive and well. (iii) *Fed.*—Birds 77, 78 and 79 used. No. 77 died in two weeks. No. 78 died in nine weeks after showing marked paralysis of the left side. No. 79 died in nine weeks without any definite symptoms or lesions.

(*d*) *Peripheral Nerves.* (1) *Injected Intra-cranially.*—Birds 80, 81, 82, 83, 84 and 85 used. No. 80 died in one week. No. 81 died in three months after showing characteristic head and neck symptoms. No. 82 died in seven weeks; nothing diagnostic noted. No. 83 became lame in five weeks, and was a typical positive case when examined after death (*see* Appendix III.). No. 84 died in three months; nothing diagnostic noted. No. 85 survived and has never shown any evidence of infection. (ii) *Injected Subcutaneously.*—Birds 86, 87, 88 used. No. 86 died in eleven days. No. 87 died in five and a-half months, but showed nothing diagnostic. No. 88 still survives. (iii) *Fed.*—Birds 89, 90 and 91 used. No. 89 died in seven days. No. 90 died in nine weeks. No. 91 died in seven weeks. These birds showed no evidence of infection as far as clinical or post-mortem appearances were concerned.

Controls (untreated birds).—Of the twenty-five control untreated birds, seven had died up to nine months during which the experiment was under observation. The cause of their death was not ascertained definitely, but in no case was there any evidence to suggested fowl paralysis as a cause, nor did any show the slightest evidence of such a condition on post-mortem examination.

The net result of Experiment II. is that we have definite evidence of having artificially infected five normal birds treated with tissues of affected cases (spleen 3, lungs 1, peripheral nerves 1), the evidence being the development of characteristic clinical symptoms during life, and the appearance of typical post-mortem lesions. If we add to this number the treated birds which showed clinical, but no definite post-mortem features, there is a total of 10 (spleen 4, lungs 2, brain 2, and peripheral nerves 2). Again, if we include those cases in which histological lesions only in the brain were found, following deaths 2–5 months after treatment, as Pappenheimer did, the number would be considerably higher. We have

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purposely omitted these in our deduction. The number of deaths during the nine months following the treatment in the treated and control birds is interesting, and we venture to say, suggestive. Of the 25 control, untreated birds, 7, or 34 per cent., died, while of the 71 treated in various ways with material from affected birds, 59, or 83 per cent., died. (Spleen, 10 out of 15, or 66 per cent.; lungs, 10 out of 12, or 83 per cent.; brain, 21 out of 23, or 91 per cent.; and peripheral nerves, 18 out of 21, or 85 per cent.) Further transmission and "passage" experiments are still in progress.

Transmission from Adult to Progeny.

Various observers have voiced the opinion that fowl paralysis is transmitted from the adult to the chick *via* the egg, and that the incubation period is two months or more. Doyle (1927) says: "We have reproduced the disease by merely breeding from an experimental flock in which there had been cases of paralysis, and in which there were cases of iritis. Several of the birds hatched from eggs laid by the affected flock developed typical paralysis with well-marked neuritis and myelitis before they were two months of age—one at forty-seven days. Control birds from a flock in which the disease was never known to have occurred have not shown evidence of neuritis or paralysis during a period of six months. . . . We have repeatedly observed the disease to occur for the first time in flocks following the introduction of birds from affected flocks. We have also observed the disease to develop in birds which were obtained as day-old chicks from affected flocks, and then raised in new houses on new ground. The observations which we have made regarding the spread of neuritis or paralysis in the field indicates that the important factor in its dissemination is the sale of breeding stock, baby chicks or eggs for hatching, from a flock in which one of several birds have shown symptoms and lesions of this disease." Marginson and McGaughey (1931) refer to fifteen outbreaks, in nine of which there was strong evidence that infection was introduced through eggs, and in six by young chicks. Commenting on their observations, they say: "We do not intend to imply that this method of spread is the only possible one, but desire to point out its importance. It would appear that the principal method of spread is through breeding from affected adult birds." Bayon (1931), referring to preventive measures, says: "Last, but not least, the source of the strain affected must be considered and scrutinised, and if necessary avoided in the future." Beaudette and Hudson (1931) state that the appearance of the disease on a new farm is always preceded by the introduction of hatching eggs, baby chicks, or breeding stock, and that it is always possible to trace new outbreaks to introduction of eggs, etc., from an infected farm. They conclude by stating that "while there is much to be done before the question of transmission can be finally settled, the evidence is strongly in favour of the conception of transmission through the egg."

We have much evidence in favour of the egg transmission theory, and have many examples typical of those quoted by the above authors. Of the thirty-

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three outbreaks of which we have detailed records, the source of the infected birds was in six bought-in eggs, in six bought-in day-old chicks, and in eleven bought-in pullets (two to six months of age), while in the remaining ten the birds were hatched from the poultry farmers' own hens bred to imported stock. Again, we have much evidence that the disease may be confined entirely to one group of birds, while others of the same age, hatched in the same incubators at the same time, and reared in the same houses and on the same land, and fed on the same food and tended by the same people, have always remained healthy (*see* Appendix I. (*d*) for an example of such an occurrence). Further, we have several records of the disease being confined to the progeny of one group of birds on a farm, that group having had cases of fowl paralysis in it during their pullet year, and we have a record of a single hen in a flock producing diseased stock (*see* Appendix II.). On one farm the disease appeared in the progeny of stock which showed cases of iritis, and no other symptoms of fowl paralysis, while in another outbreak there was at least 30 per cent. of cases of iritis, and some lameness among the breeding stock. It would thus seem that our field observations on transmission of fowl paralysis through the egg agrees closely with those of the above authors. We feel that the conclusion from such observations, namely, that egg transmission does take place, is further justified from the fact that ovarian tumours composed of typical fowl paralysis cells are common. (We have records of fifteen in seventy-six outbreaks, *i.e.*, nearly 20 per cent.) While we have insufficient evidence to justify a conclusion that the male bird may play a part in transmission, there are good grounds for believing that this may occur in some cases. (In ten out of thirty-three outbreaks the only imported stock were cockerels.)

We have had the opportunity of carrying out a laboratory experiment in connection with egg transmission. From a heavily infected flock three consignments of eggs were obtained and hatched by us in our own incubators. The chicks were subjected to various conditions (as detailed in Appendix IV); in six birds typical fowl paralysis developed. This carefully controlled experiment adds more evidence to support the contention that fowl paralysis may be egg transmitted, and that the association of intestinal parasites is not essential.

Transmission from Affected to Normal Stock by Contact.

Evidence is accumulating to support the view of some observers that fowl paralysis may be transmitted under natural conditions by simple contact. Marginson and McGaughey (1931) produced evidence that this takes place in the field. Doyle (1927) observed the disease to occur in a control bird after it had been associating intimately with iritic and neuritic birds for eleven months. Emoto and Miyamoto (1930) observed fowl paralysis in Japan, and state that "in the farms where affected birds have once appeared, the disease occurs every year afterwards. Not only the imported birds and their offspring, but also ordinary hens of our country have come to be affected by this disease, *i.e.*, this disease can be also considered from its epidemiology to be an infectious disease."

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In our records we have but little evidence that contact may cause infection, but in our own experimental stock we found a typical case in a hen which had been housed in a pen adjacent to one housing many fowl paralysis birds. The same attendant was in charge of both pens. It would appear from such evidence that spread by contact of affected and healthy birds is possible.

Discussion.

While many forms of paralysis may attack poultry, and the causes of such are known or suspected, we believe that there is a specific condition at present termed fowl paralysis. (This name is, in our opinion, a misnomer, in that, although paralysis is a prominent symptom, cases do occur in which there is no suggestion of paralysis. We favour the name lymphomatosis—omitting the prefix "neuro-".) From our observations we believe we can readily diagnose the condition, especially in an outbreak in which a number of birds are affected. We rely on clinical symptoms, post-mortem lesions, and the histological picture of nervous and other tissues. It may be difficult at times to diagnose the condition by the examination of a single bird, especially if indefinite symptoms only are presented. Again, the histological picture of various areas of the brain and spinal cord may be misleading in that the number of typical cells (lymphocytes, plasma cells, and mononuclears) may be small, and may be confused with the appearance of certain cells in the brain, etc., of so-called normal birds. As a rule, however, when such typical cells are found they are readily recognised. When a doubt exists we find it better to obtain fresh specimens for examination, or, better still, to examine the entire flock from which the affected birds came, when ample evidence of the existence of fowl paralysis will usually be found.

Much has been written on the causation of this disease, and agents as wide apart as intestinal parasites, lack of essential vitamins, etc., in the diet, and an ultra-visible virus as the cause, all have their supporters. We have examined the various theories, and in the light of our field and laboratory experiments and observations, we feel justified in concluding that neither of the first two agents is the true cause. We have successfully transmitted the disease by the injection of bacteriologically sterile tissues (spleen, lungs, and nervous tissue). We have made no experiments with emulsions of tissue passed through bacterial filters. We agree that in many affected cases coccidia and other intestinal parasites are found, and in some cases in considerable numbers, but in many other affected birds, and in fact in many outbreaks, no trace of such parasites can be found. We are not convinced that these parasites are either primary or secondary to fowl paralysis infection, for the disease can be reproduced artificially in the presence or absence of such parasites. It has been stated that the elimination of coccidia, tapeworms, etc., from a stock, or the feeding of vitamins in suitable form, will effect a cure, or will prevent the occurrence of further cases of fowl paralysis. We cannot confirm this observation (as an example, *see* Appendix I. (*a*)). It is evident from our experiments that suitable tissues of affected birds possess some factor capable of setting up symptoms and lesions diagnostic of

fowl paralysis when injected into healthy birds, and that it can be further transferred from these artificially infected birds to at least one further series. The incubation period of the naturally occurring and the artificially produced disease is also of significance; it is usually between two and six months. The whole epidemiology of the condition strongly suggests that the cause is specific, though it seems probable that secondary influences, *e.g.*, coccidia, tapeworms, errors in feeding and in-breeding, have some effect on its incidence. The individual case in the flock is always puzzling, but can probably be explained on the egg-transmission theory, coupled with the influence of the above secondary factors. We also feel justified in concluding from our observations and experiments that the disease is probably egg-transmitted, and that the chief source of spread is the importation of eggs for hatching, day-old chicks, pullets, or cockerels from affected stock. We desire to point out that in many cases it is well-nigh impossible to diagnose the condition in some breeding stocks whose progeny develop fowl paralysis, and thus on some occasions the poultry breeder is entirely ignorant of the fact that his breeding stock is a source of infection; it is not till the progeny exhibit definite symptoms, when two to six months of age, that he may suspect latent infection in the parent birds. Again, it seems evident that the subsequent treatment of the progeny of such stock may play a big part in the production of typical cases of the disease. Under ideal rearing conditions, etc., it seems probable that the disease may remain latent even for some generations. The hen with typical fowl paralysis ovarian tumours would seem to be bound to transmit infection to her chicks, and though absolute evidence seems wanting, it is highly probable that the male bird acts also as a transmitter. Because of the latent nature of the disease, it will always be difficult to prove absolutely that transmission by contact takes place under natural conditions. The appearance of the disease among the birds native to Japan following the importation of diseased foreign stock is highly suggestive of contact transmission.

We know of no definite curative or preventive measures save the elimination of the diseased (patent or latent) breeding stock, and we feel that the various methods advocated are palliative in that they merely mask the existence of the disease by preventing or retarding the development of symptoms in the treated birds, and so tend to allow the condition to be carried over into the next or subsequent generations.

Summary and Conclusions.

(1) Fowl paralysis exists as a specific disease, and can be diagnosed by a study of clinical symptoms, post-mortem lesions, and histological sections of various tissues, including the brain and spinal cord. (2) It is more readily diagnosed as an outbreak than by the examination of a single affected bird. (3) The disease can be transmitted to healthy birds by the injection of suitable sterile tissues from affected cases. (4) The disease may exist without the presence of intestinal parasites, including coccidia, and can be reproduced in birds which have never been exposed to such parasitic infestation. (5) It seems probable that intestinal parasites, errors in feeding and in-breeding, predispose to the production of

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symptoms. In the absence of the causal factor (at present unknown, but contained in tissues of affected birds) they are incapable of causing evidence of true fowl paralysis. (6) It seems practically certain that the disease may be transmitted through the egg, and that the cockerel as well as the hen may be the transmitter. (7) There is some evidence to show that transmission by contact between diseased and healthy birds may occur. (8) The hypothesis that the cause of the disease is a filtrable virus is consistent with our results.

Appendix I.

Four Examples of Typical Outbreaks of Fowl Paralysis.

(*a*) In August, 1930, we were consulted with regard to a seemingly obscure disease among a flock of fowls. The outbreak was characterised by lameness, iritis, drooping wings, swollen crops, diarrhœa, and general paralysis, and the death-rate had been heavy. We did not recognise the disease as fowl paralysis for some months, and in the meantime advised treatment for intestinal parasites, among which we demonstrated the presence of coccidia, tapeworms, and various varieties of round worms. All the usual remedies were tried as curative and preventive measures, including the injection of carbon tetrachloride *via* the rectum, without any decrease in the incidence. Cod-liver oil and vitamins in a concentrated form were then used on some groups, and in others a liberal ration of milk was fed. No good results were found. As the colder weather developed the incidence increased. By this time we were fairly certain that we were dealing with an outbreak of fowl paralysis. (The description of the disease given by us in "Eggs" (1931) concerned this outbreak.) Many post-mortem examinations have been made, and lesions of all types found, including ovarian tumours in 16.6 per cent. of the birds examined. The disease has continued on the farm for over twelve months, affecting all birds except one lot of Light Sussex obtained from an outside source. They have been run with the affected, and treated alike and tended by the same staff. The affected stock were all bred by the owner from a stock he has bred for many years; cockerels only were imported annually. Birds up to two years old have developed the typical disease, but it was first seen in pullets. The only indication of the existence of the disease in the flock previous to 1930 was the appearance of a few lame and blind birds on occasion. The loss has been extremely heavy.

(*b*) A poultry keeper purchased eggs from a breeder, hatched chicks from them in an incubator, and reared the chicks to the age of thirteen weeks in batteries. Some birds were then sold to another poultry keeper. We found unmistakable evidence of fowl paralysis within a fortnight in these birds. At least 10 per cent. of the birds showed symptoms. The remainder were sold, and the farm restocked. As far as we know, the second lot of birds have done well. We now examined the remainder of the stock retained by the seller, and which were still in batteries. We found unmistakable evidence of the existence of fowl paralysis, but not to such a marked extent as in those sold. This is interesting in that many outbreaks seem to coincide with the transferring of birds from batteries or brooders to a

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free range or a semi-intensive life. We have no direct evidence that the original breeding stock showed any evidence of infection, but we have some proof that the stock at one time produced progeny, which in their turn produced diseased pullets.

(*c*) From a reputedly clean stock the nucleus of a new stock on a new poultry farm was acquired. For several years no sick birds were found among the stock or their progeny. Each year fresh stock cockerels from outside sources were added to the farm. During the present year we have confirmed fowl paralysis among birds hatched from eggs produced by that stock. In one case only one or two cases were found, but in the other the incidence was much higher. It should be stated that the original stock is kept under ideal conditions, while the infected stocks are in the possession of amateur poultry keepers, whose method of of rearing, etc., are not of the best. It seems possible that the original stock harbours latent infection, which is masked by the system of management, and that the conditions under which the progeny have been reared has caused the infection to become patent.

(*d*) A poultry keeper starting poultry farming bought 3,000 sex-linked, day-old chicks from a hatchery, and 500 White Wyandotte day-olds from another source. These birds were kept in the same brooders for seven weeks, and were then allowed free range on land on which poultry had never ran, and had ample allowance of space per bird. Typical fowl paralysis appeared among the sex-linked, while the White Wyandottes have always remained perfectly healthy. It seems evident that the disease existed solely in the stock producing the sex-linked chicks, and that the White Wyandottes were from "clean" stock.

Appendix II.

EXAMPLE OF ONE BIRD IN A FLOCK PRODUCING AFFECTED PROGENY.

In a flock of Rhode Island Reds was hen 115, which had an excellent egg record. She was bred, and seventy-four of her eggs were hatched and yielded fifty-nine chicks. Between two and five weeks after hatching several died from an undiagnosed cause. Fourteen of the remainder were rung and therefore easily recognised; of them, six died at ages between five and seven months. No other birds have died on the farm during the present season. We had opportunity of examining four of the six birds. In two there were swollen nerves with the usual microscopic picture. One had a typical ovarian tumour, and in the other was a huge tumour under the skin of the neck. The cells composing these tumours were typical of fowl paralysis. It is of interest to note that in no case was there any evidence of disease of the duodenum, and the only parasites found were a few coccidia in the cæca of one bird. It seems evident that the disease was transmitted by hen 115, and not by the male birds, as none of the progeny of any other hen has shown any signs of disease.

Appendix III.

DETAILS, *re* ARTIFICIALLY INFECTED BIRDS.

No. 5 (White Spiral).—Injected intravenously with lung emulsion. Became lame in the right leg three months after injection. By the eighth day the lameness

had so progressed that the leg was simply dragged behind the bird. The left wing was drooping. *Post-mortem*.—Much duodenal catarrh and inflammation. No coccidiosis or any other intestinal parasite. Right sciatic nerve and lumbar-sacral plexus much swollen. Left brachial nerve much swollen. Typical cells in nerves and plexuses.

No. 21.—Injected intramuscularly with mixture of spleen and lungs from No. 5. Six months after injection bird began to gasp and became completely prostrate, lying on its left side, It died within twenty-four hours. *Post-mortem*.—Some duodenal catarrh. No intestinal parasites. Nerves and plexuses apparently normal. Tumours present in both lungs and in pancreas. Typical cells in tumours.

No. 23.—Injected intravenously with spleen emulsion. In two months and nine days after injection gasping observed, followed by lameness, and subsequently general paralysis. *Post-mortem*.—Left sciatic nerve and lumbo-sacral plexus much swollen. Typical cells in both tissues. No abnormal condition of duodenum. A few coccidia in the cæca only.

No. 26.—Injected intramuscularly with spleen emulsion. In eight weeks after injection bird became lame and showed a drooping wing. The lameness increased during the next nine days, when the bird was killed accidentally. *Post-mortem*.—The carcase had been badly mutilated, but we were able to note that the one lumbo-sacral plexus was much swollen. Typical cells were easily demonstrated.

No. 51.—Injected intravenously with spleen emulsion. Nine weeks after injection the bird developed a typical drooping wing. *Post-mortem*.—The right brachial plexus was much swollen, and the left slightly so. Both showed typical cells. A few coccidia only were present in the cæca.

No. 58.—Injected subcutaneously with lung emulsion. This bird showed evidence of paralysis thirteen weeks after injection. *Post-mortem*.—Right brachial plexus swollen and contained typical cells. No marked changes in the duodenum. A few coccidia and round worms in the cæca.

No. 83.—Injected intra-cranially with emulsion of peripheral nerves. Showed lameness in four and three-quarter months. Still lame at five months ; lameness decreased till the bird died a fortnight later. *Post-mortem*.—Brachial plexus swollen. Slight duodenal catarrh. A few coccidia of the *Eimeria maxima* type in the duodenum.

Appendix IV.

Details of Egg Transmission Experiments.

Three lots of eggs produced by heavily infected stock were received by us, and from them we hatched out three lots of chicks (*a*), (*b*) and (*c*). Lots (*a*) and (*b*) were run together, while lot (*c*) was kept separate from the others.

Lots (*a*) and (*b*) were reared in a new house with sun parlour and wire floor, and were later transferred to a grass run. One chick died seven days after hatching (nail in gizzard) ; two died at ten days (one B.W.D., one Aspergillosis) ; two died

at four weeks (coccidia found in the cæca). The remaining chicks were then treated as under: (1) When two and a-half months old, eighteen were moved to a new house with a grass run and were fed coccidia. Blood was found in the droppings ten days later, but none died. All have remained healthy to date; they are now seven months old. Two were recently killed and examined; no parasites or abnormal conditions were found. (2) When two and a-half months old, twenty were vaccinated with pigeon-pox vaccine. (This was done because it was soon after such treatment that an outbreak of fowl paralysis occurred in a flock of birds of which we have a record.) All "took" well. All have remained well. Two birds killed for post-mortem work yielded results as in (1). (3) When three and a-half months old thirteen were placed in small cages, and were kept there for three months. All are alive to-day, and none has shown any symptoms of disease. (4) When three and a-half months old, twenty were sent to the original farm and placed on infected land. In three months one died, and the post-mortem examination showed swelling of the sciatic nerves and many coccidia, round worms, and tapeworms in the duodenum. Three cockerels have shown lameness, and have apparently recovered. (5) The remainder have been kept without any treatment or movement. One bird went lame and recovered. One bird gasped when five months old, and died seven days later after showing typical head and neck symptoms. The post-mortem showed typical tumours in both lungs. No intestinal parasites of any kind were found. A second bird was killed, and showed typical liver lesions, while two birds have recently developed a typical drooping wing. The others are healthy.

Lot (*c*) were reared on a grass run from day-olds. At five months one was inclined to topple over, and was killed, but no definite lesions were found, nor were any intestinal parasites observed. All the others are healthy, except two which have developed "eye" symptoms.

We desire to tender our best thanks to Dr. G. M. Findlay, who very kindly cut for us, and examined a number of sections.

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Clinical Articles

Pericarditis of the Fowl.

By K. D. DOWNHAM, B.V.Sc., M.R.C.V.S., D.V.H.,
Advisory Veterinary Officer, Harper Adams Agricultural College, Newport, Salop.

THE above condition is commonly met with in poultry where large numbers of post-mortems are made. A brief description of this affection may, therefore, be of some value to those who are interested in diseases of the fowl. Inflammation of the pericardium may result from a general blood infection; it may also arise through the spreading of infection from the surrounding parts, or from local irritation. Among the first class of cases, leukæmia, gout, and rheumatic conditions generally, appear to be the most frequent causes of the disease as it affects the fowl. It is commonly seen in birds suffering from nephritis, avian tuberculosis, septicæmia, and in many toxæmic conditions. When the disease arises from local causes, neoplasms are most frequently responsible.

In pericarditis arising from a general blood infection, the first noticeable change of the pericardium is its increased vascularity: the smooth glossy surface disappears, and it becomes injected with a fine network of blood vessels. Exudation of lymphocytes and fibrin takes place, and these are deposited upon the membrane. The two opposed surfaces of the pericardial sac separate; serum is formed; the fluid is generally amber-coloured, but may be turbid from the accumulation of corpuscles and pus cells. Sometimes a considerable amount of sero-fibrinous fluid is formed: this separates the two layers of the pericardium, and allows lymph processes to stretch from layer to layer; in time the fluid is absorbed or organisation takes place, and the parietal and visceral layers of the pericardium become united with connective tissue. The amount of fibrous tissue laid down varies in different cases: in some birds the pericardium resembles thick parchment, in others a gelatinous appearance is seen. In avian tuberculosis the tuberculous nodules are found in the organised tissue and upon the original membrane which covers the heart's surface.

Cohnheim has shown that the higher the tension in the pericardium the smaller is the output from the heart. It is observed that birds suffering from pericarditis are generally emaciated or suffering from debility: these conditions are accentuated by the impaired action of the heart.

The photograph (*see illustration facing p.* 6) shows a fowl suffering from pericarditis with effusion. There was a considerable amount of sero-fibrinous fluid present in the heart sac: this is clearly shown in the illustration.

In am indebted to Mr. Morley Davies, M.A., of this College, for kindly photo-graphing the specimen.

Abstracts of Current Literature

L. de Blieck.—Poultry Diseases at the Institute for Parasitic and Infectious Diseases, Utrecht. *Tijdschrift voor Diergeneeskunde*, 1931, Deel 58, Afl. 18, No. 153.

F. A. DE ZEEUW summarises the various poultry diseases in Holland, and draws special attention to such diseases as cause the death of poultry. The percentage of 1,737 hens submitted to the institute for post-mortem examination show that 17.2 were affected with salpingitis, accompanied by peritonitis; 8.5 pullorum infection; 8.5 coccidiosis; 6.7 leucæmia; 6.5 intestinal worms (davainea, heterakis, ascaridia, capillaria); 4.6 neurolymphomatosis gallinarum (polyneuritis); 5.5 fowl-pox (diphtheria); 5.9 coryza infectiosa (contagious catarrh); 5.7 tuberculosis; 6.5 metabolic diseases, especially gout; 3.2 hæmorrhages without a specific cause (in most cases of hepatic origin); 6.8 fowl cholera; 5.8 enteritis; 6.7 tumours, especially sarcomata; 1.7 fowl typhoid (Klein's disease).

Dr. H. S. Hofkamp, of the Laboratory of Tropical Diseases in the Institute of Parasitic and Infectious Diseases, summarises his paper on cross experiments with the pigeon coccidium and the fowl coccidium. Four of eleven chickens were successfully infected by the mouth with *Eimeria Pfeifferi*. Healthy chickens were then given oöcysts obtained from the fæces of these four chickens. A serious infection resulted. After one passage through chickens, the pigeon coccidium appeared to have become as virulent for other chickens as the ordinary fowl coccidium *Eimeria avium vel tenella*.

Of four young pigeons which received by the mouth fowl coccidia (*E. avium*), two contracted a slight infection. Morphologically, fowl coccidia and pigeon coccidia are identical, except that there is a slight difference in the shape of the oöcysts. After development in chickens the oöcyst of the pigeon coccidium takes the form of fowl coccidia. The author is of the opinion that the pigeon coccidium, *Eimeria Pfeifferi* (Labbé), and the fowl coccidium, *E. tenella* (Railliet and Lucet), do not belong to different species, but that they are identical.

As a chemical disinfectant, caporit (a proprietary preparation of lime hypochlorite) in a 10 per cent. solution proved to be effective. The oöcysts were destroyed very quickly. A 10 per cent. solution of sodium or of antiformin was found to be useless. Therapeutically, the author tried creolin, tricresol, spirocid, Rivanolette, igitol, and ichthargan. Only ichthargan ($\frac{1}{3}$ gramme per kilogramme) gave satisfactory results. Three plates are given with the article. (*Tijdschrift voor Diergeneeskunde*, 1931, del 158, Aflevermig 16, No. 154.)

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Correspondence

DISTEMPER IMMUNISATION.

To the Editor of THE VETERINARY JOURNAL.

SIR,—My attention has been drawn to an editorial note which appeared in THE VETERINARY JOURNAL of December, 1931, entitled "Distemper Immunisation by Dried Virus." This title appears to be most unfortunate, as, in my opinion, it will tend to confuse the veterinary practitioner, as it does not mention either vaccine or immune serum, one of which must be employed in combination with the virus in the production of immunity.

Whilst full acknowledgments have been given by Dr. Laidlaw and Mr. Dunkin to the brilliant pioneer work of Carré and Puntoni, it must be realised that the French and Italian work was abandoned in an unfinished state owing to the authors having no efficient organisation for the supply of susceptible animals. Dr. Laidlaw and Mr. Dunkin therefore had to start their research from the beginning, and brought it to a very successful and convincing stage by a field experiment in which several thousand hounds and dogs were immunised against distemper.

It remained for Major Dalling to organise the production of vaccine and virus and immune serum on a manufacturing scale, and I feel certain that everyone who realises the difficulties of switching from laboratory to commercial production will heartily congratulate him on bringing his anxious and laborious task to the point of consistent success. However, I must point out in this connection that the method of drying virus, the degree of dryness that must be attained, and the keeping qualities of such a product were the outcome of research by Dr. Laidlaw and Mr. Dunkin, and that full details of their work was published in the *Journal of the American Veterinary Association* of April, 1931. The paper was received for publication on January 28th, 1931. The experiments must, therefore, have been carried out in 1930, or earlier, as many of them required months for their completion. During the latter part of this period, as stated in his paper, Major Dalling has been in close and friendly co-operation with Dr. Laidlaw and Mr. Dunkin, and has succeeded in producing on a commercial scale an exceedingly satisfactory dried virus by following the principles laid down by Dr. Laidlaw and Mr. Dunkin.

I am writing this, as the editorial note seems to me to convey a very erroneous impression of the facts of the case, which I have no doubt is in every way contrary to your wish.—I am, Sir, yours faithfully,

S. R. DOUGLAS, F.R.S., *Chairman,*
Distemper Research Committee.

National Institute for Medical Research,
Hampstead, N.W.3.

DISTEMPER IMMUNISATION BY DRIED VIRUS.

To the Editor of THE VETERINARY JOURNAL.

SIR,—It has been pointed out to us that the Editorial in your December number may give rise to some misunderstanding. Probably owing to the necessity of editorial compression, you did not say at length what is obvious to those of your readers who have followed the history of distemper prophylaxis in the veterinary press. If you had written a fuller article, you would doubtless have repeated the oft-told story

that the method of immunisation with vaccine and virus, discovered and worked out in detail by Laidlaw and Dunkin, has never failed in the laboratory work at Mill Hill; or in the work we undertook here after the Mill Hill workers had published their results and kindly made all details available to us in personal discussion. As we have stated earlier, we have abundantly confirmed this discovery.

So far as field work is concerned, Laidlaw and Dunkin immunised many packs of hounds, with the success described in their article in the April number of the *Journal of the American Veterinary Association*. Immediately after their early field work, we also immunised in the field about 2,000 dogs, with, so far as we can ascertain, the same success; thus further confirming their work. The commercial issue was then taken up, and we issued vaccine and virus for the immunisation of some 20,000 dogs. In the summer of 1930, after some disturbing reports, we were able to prove, in controlled laboratory experiments that some at least of these dogs did not become immune; and the same observation was made by Laidlaw and Dunkin. Lengthy investigation established that some of the emulsion of virus sent out did not withstand transport, and was either reduced in potency, or dead when injected into the dogs. So far as we can trace, this accident happened in but a small percentage of instances, but as soon as we had established the facts, Messrs. Burroughs Wellcome & Co., who undertake issue, withdrew all material.

The difference of experience between Laidlaw–Dunkin and ourselves concerns the practicability of issuing "broadcast," virus in the form of emulsion, *i.e.*, "wet virus." They, working under their own conditions, and making the injections in many instances themselves or through a very small group of veterinary surgeons rigidly following their methods, succeeded in immunising large numbers of dogs; we, issuing "wet virus" during the commercial period in any quantity demanded and at any time, to veterinary surgeons in Britain, not all as familiar as the Mill Hill team with the process of immunisation and the necessary safeguards, failed in a small number of cases to get in the field the same degree of success which Laidlaw and Dunkin achieved. (So far as we know, none of the American laboratories has found it practicable to issue *wet* virus on the large scale commercially.)

We thereupon came to the conclusion that unless we could make with regularity and on a large scale, *stable* dried virus, commercial issue was impracticable. Knowing that Laidlaw and Dunkin had successfully dried virus at times during their earlier work, we concentrated all our resources on making *stable* dried virus. After several months of continuous work we succeeded in our aim, and we have, since September, 1930, issued some thousands of doses of dried virus for use in the vaccine-virus method with, so far as we can ascertain, an approach to 100 per cent. success. —We are, yours faithfully,

R. A. O'BRIEN.
THOMAS DALLING.

The Wellcome Physiological
Research Laboratories, Beckenham.

News

THE ROYAL AGRICULTURAL SOCIETY OF ENGLAND. ELECTION OF PRESIDENT.

THE election of Lord Mildmay of Flete, by the unanimous vote of the Council, as President of the Royal Agricultural Society of England, will be received with pleasure by the members of the veterinary profession, with whom his name is well known as one who takes an active interest in scientific research into the diseases of

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animals. In accepting the honour, Lord Mildmay, who is a Governor not only of the Royal Veterinary College, but also of Middlesex Hospital, and a member both of the Medical Research and the Field Distemper Councils, alluded to the importance of the work of the Society in many branches of Agricultural Science, and stated: "I have learned how interdependent is research work upon human and animal ills, and I have also learned how illimitable is the field for research."

The coming year will be an especially strenuous one for agriculture, and this means for veterinary science as well, for the progress of the two must always go hand-in-hand. This applies not only in laboratory research, but also from the point of view of the practitioner; as when agriculture is depressed the financial support of each is lessened, and, unfortunately, both depend very largely in Great Britain upon private and individual effort.

The Royal Agricultural Society has always recognised this, not only by giving its moral support to veterinary questions, but by the voting of large sums of money annually to veterinary research.

The profession appreciate very greatly their powerful and friendly support.

Obituary

DEATH OF JOHN VENNERHOLM.

It is with the greatest regret that our readers will learn of the death (on December 30th) of Professor John Vennerholm, Emeritus Professor of Surgery and a former Rector of the Stockholm College, and literally the "Grand Old Man" of the veterinary profession in Sweden. Clever to a degree and modest to a fault, his quiet earnestness and willingness to help made everybody his friend, and there is no doubt that it is largely due to the quiet work of John Vennerholm that the veterinary profession in Sweden occupies the high position which it holds at the present time in that country. His writings, especially his text-book on the Surgery of the Domesticated Animals, will always remain as permanent testimony to his name; and although of recent years one had not seen his name in Congress lists, there was a time when his name had only to be mentioned amongst veterinary colleagues for those present to at once recognise that in Professor Vennerholm the Swedish veterinary profession had a representative whose master's skill was recognised, and whose name was received with approbation.

Publishers' Notices

All communications should be addressed to 7 & 8, Henrietta Street, Covent Garden, London, W.C. 2. Telephone: Temple Bar 4646. Telegrams: "Baillière, Phone, London."

Letters for the JOURNAL, literary contributions, reports, notices, books for review, exchanges, new instruments or materials, and all matter for publication (except advertisements) should be addressed to the Editor.

Copy of advertisements should be in the hands of the publishers—Baillière, Tindall and Cox—not later than the 25th of the month, or if proof is required, not later than the 23rd.

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